CLINICAL AND BIOCHEMICAL EVALUATION OF EFFICACY OF CALCIUM FLUORIDE AS AN ADJUNCT TO NON-SURGICAL PERIODONTAL THERAPY – A RANDOMIZED CLINICAL TRIAL

A Dissertation submitted in

partial fulfillment of the requirements

for the degree of

MASTER OF DENTAL SURGERY

BRANCH – II

PERIODONTICS

THE TAMIL NADU DR. M.G.R. MEDICAL UNIVERSITY

Chennai – 600 032

2014 - 2017

CERTIFICATE BY HEAD OF THE DEPARTMENT /

HEAD OF THE INSTITUTION

This is to certify that the Dissertation entitled **"CLINICAL AND BIOCHEMICAL EVALUATION OF EFFICACY OF CALCIUM FLUORIDE AS AN ADJUNCT TO NON-SURGICAL PERIODONTAL THERAPY – A RANDOMIZED CLINICAL TRIAL"** is a bonafide work done by **Dr. ANJU M K**, Post Graduate student (2014–2017) in the Department of Periodontics, under the guidance of **Dr. K MALATHI**, HOD and Professor (Guide), Department of Periodontics, Tamil Nadu Government Dental College and Hospital, Chennai – 600 003.

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DECLARATION BY THE CANDIDATE

I hereby declare that this dissertation titled **"CLINICAL AND BIOCHEMICAL EVALUATION OF EFFICACY OF CALCIUM FLUORIDE AS AN ADJUNCT TO NON-SURGICAL PERIODONTAL THERAPY – A RANDOMIZED CLINICAL TRIAL"** is a bonafide and genuine research work carried out by me under the guidance of **Dr.K MALATHI. M.D.S.,** HOD and Guide, Department of Periodontics, Tamil Nadu Government Dental College and Hospital, Chennai -600003.

Dr. ANJU M K

Signature of the candidate

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TRIPARTITE AGREEMENT

This agreement herein after the "Agreement" is entered into on this day ----------between the Tamil Nadu Government Dental College and Hospital represented by its **Principal** having address at Tamil Nadu Government Dental College and Hospital, Chennai – 600 003, (hereafter referred to as, 'the college')

And

Dr. ANJU M K, aged 30 years currently studying as **Post Graduate student** in Department of Periodontics, Tamil Nadu Government Dental College and Hospital, Chennai – 600 003, (hereafter referred to as 'the PG student and principal investigator')

And

Mrs. Dr .K MALATHI aged 45 years working as **HOD and Professor** in Department of Periodontics, Tamil Nadu Government Dental College and Hospital, Chennai and **Dr. V S PARTHIBAN, BHMS**, working as Asst. Medical Officer (Homeopathy) , Govt. Headquarters Hospital, Tiruvallur (herein after referred to as the 'Co- Investigators'),

Whereas the PG student as part of his curriculum undertakes this research on **"CLINICAL AND BIOCHEMICAL EVALUATION OF EFFICACY OF CALCIUM FLUORIDE AS AN ADJUNCT TO NON-SURGICAL PERIODONTAL THERAPY – A RANDOMIZED CLINICAL TRIAL"** for which purpose the Co-investigators and the college shall provide the requisite infrastructure based on availability and also provide facility to the PG student as to the extent possible as a principal investigator.

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In witness whereof the parties hereinabove mentioned have on this day month and year herein above mentioned set their hands to this agreement in the presence of the following two witnesses.

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Sub: IEC review of the research proposals,

Title of the work: Clinical and Biochemical evaluation of efficacy of calcium fluride as an adjunct to non-surgical periodontal therapy – A randomized clinical trial.

Principal Investigator: Dr. Anju M. K.
II Yr. M.D.S., Student.

Department : Department of Periodontics
Tamil Nadu Govt. Dental College & Hospital , Chennai-3

Thank you for submitting your research proposal , which was considered at the Institutional Ethics Committee meeting held on 02-07-2015, at TN Govt. Dental College and the documents related to the study referred above were discussed and the modifications done as suggested and reported to us through your letter dated 23-09-2015 have been reviewed.

The decision of the members of the committee , the secretary and the Chairperson IEC of TN Govt. Dental College is here under:

Approved	Approved and advised to proceed with the study
Approved with suggestions	----------
Revision	----------
Rejected	----------

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LIST OF ABBREVIATIONS

OPN	**Osteopontin**
Calc-f	Calcarea fluor
Ca^{2+}	Calcium
RANKL	Receptor activator of nuclear factor kappa B ligand
OPG	Osteoprotegrin
RANK	Receptor activator of nuclear factor kappa B
BOP	Bleeding on probing
PI	Plaque index
GBI	Gingival bleeding index
PPD	Probing pocket depth
CAL	Clinical attachment level
PAL	Probing attachment level
SRP	Scaling and root planning
MWF	Modified Widman Flap
CEJ	Cemento-enamel junction
ELISA	Enzyme-linked immunosorbent assay
OD	Optical density

Introduction

INTRODUCTION

Periodontitis is a chronic infectious disease, triggered by the host immune response to an array of periodontal biofilm-associated microorganisms, which ultimately leads to the inflammation of the tissues supporting the teeth.

The initial host response to bacterial infection is a local inflammatory reaction that activates the innate immune system. An imbalance between biofilms and immune system results in over expression of an array of pro-inflammatory cytokines, propagation of inflammation through the gingival tissues and subsequent destruction of alveolar bone. Thus, the inflammatory process results in destruction of connective tissue and alveolar bone – ***Hallmark of periodontal disease.***

Diagnosis of periodontal disease has been primarily based upon clinical and radiographic measures of periodontal tissue destruction. These parameters provide a measure of past destruction and are of limited use in early diagnosis ***(Frodge et al, 2008)***[1]. Biomarkers of disease play an important role in life sciences and have begun to assume a greater role in diagnosis, monitoring of therapy outcomes, and drug discovery ***(Teles et al, 2010)***[2]. For biomarkers to assume their rightful role in routine practice, it is essential that their relation to the mechanism of disease progression and therapeutic intervention be more clearly understood.

One of the biomarkers of alveolar bone destruction is osteopontin. Osteopontin (OPN) is a non-collagenous, calcium binding, glycosylated phosphoprotein produced by osteoblasts ***(Rodan, 1994)***[3]. Osteopontin level reflects active lesions of aggravated periodontal disease accompanied by alveolar bone resorption. The concomitant increase of osteopontin in plasma is caused by spillage or overflow of osteopontin from the diseased periodontal tissues or produced by circulating activated macrophages. The level of

osteopontin before and after treatment can be used to assess the severity of periodontal disease.

Treatment of periodontal disease includes both non-surgical and surgical therapy. Non-surgical therapy remains the corner stone of periodontal treatment. It includes scaling and root planing under local anaesthesia to provide patient comfort and to control the disease progression. Systemic therapy may be employed as an adjunct to local measures and for control of systemic complications.

Homeopathy is a system of alternative medicine that was introduced by German physician and scientist Dr Samuel Hahnemann in 1796. It is based on the principle of '*similia similibus curentur'* (like cures like) ***(Lessell, 1995)[4].*** This means that the medicines used are derived from substances that if taken by a healthy person can cause symptoms similar to the illness, but when taken by the patient they trigger the body's natural healing process. Homeopathy is recognised by the World Health Organization (WHO) as the second largest system of medicine in the world today. The medicines used can be in both low and ultra-molecular dilution but both clinical experience and evidence suggest that they are still effective.

Homeopathic remedies are based on a holistic approach to health that addresses illness by stimulating the body's own healing powers. Some investigators have proved that homeopathic drugs can be used as an adjunct to nonsurgical therapy in dentistry. So, the present study is to investigate the effectiveness of homeopathic drug, calcarea fluor, as an adjunct to non-surgical therapy in the treatment of chronic periodontitis.

Aim and Objectives

AIM

To evaluate the efficacy of calcarea fluorica (30c) as an adjunct to non-surgical periodontal therapy .

OBJECTIVES

- To compare the clinical parameters before and after the treatment in each group.
- To compare the clinical parameters between different groups.
- To compare osteopontin level in plasma in each group before and after treatment.

Review of Literature

REVIEW OF LITERATURE

Periodontal disease is characterized by tissue inflammation and destruction of the tooth supporting structures that eventually leads to the loss of affected teeth ***(Kinane 2001, Page & Kornman 1997, Philstrom et al. 2005)***[5,6,7]. Despite our increased understanding of the etiology and pathogenesis of periodontal infections, the diagnosis of these diseases is still based almost entirely on traditional clinical assessment ***(Armitage 1995)***[8]**.** To arrive at a periodontal diagnosis, the dentist must rely upon factors such as (a) presence and absence of signs and symptoms, including pain, ulceration and amount of observable plaque and calculus (b) patients medical and dental history (c) presence or absence of clinical signs of inflammation like bleeding on probing (d) probing depths and (e) extent and pattern of clinical attachment and bone loss. These parameters provide a measure of past destruction and are of limited use in early diagnosis ***(Frodge et al,2008)***[1]**.** Because of the increasing prevalence and associated comorbidities, screening and diagnostic modalities for the early identification of periodontitis, its initiation and progression, as well as objective measures for response to therapy, are being sought. ***(Beck et al 2005, Genco et al 2001, Seymour et al 2007, Mealey & Oates 2006)***[9,10,11,12]**.**

Osteopontin:

A periodontal diagnostic tool provides pertinent information for differential diagnosis, localization of disease, and severity of infection. These diagnostics, in turn, serve as a basis for planning treatment and provide a means for assessing the effectiveness of periodontal therapy ***(Taba et al 2005)***[13]**.** In the disease process, potential biomarkers of the disease activity would need to be involved in some way in the disease process or

released as a consequence of tissue damage during disease progression. A biomarker is an objective measure that has been evaluated and confirmed either as an indicator of physiologic health, a pathogenic process, or a pharmacologic response to a therapeutic intervention.

Osteopontin (OPN)- a bone related biomarker, is a non-collagenous, calcium binding, glycosylated phosphoprotien produced by osteoblasts. OPN is important in immune activity and bacterial resistance and was named early T-lymphocyte activation 1 protein (Eta-1) by ***Patarca et al in 1989***[14]. Today the protein is designated as OPN and is known to be involved in bone resoption, wound repair, immune function , cell survival and cancer biology.

Bone remodelling is a regulated process in which removal of old bone by osteoclasts is followed by bone formation by osteoblasts. OPN influences bone hemostasis both by inhibiting mineral deposition, by promoting differentiation of osteoclasts and by enhancing osteoclastic activity. ***(Standal et al 2004)***[15]**.**

Bone matrix consists of an inorganic component in the form of hydroxyapatite and an organic component consisting of proteins and proteoglycans. OPN is a major non-collagenous protein in bone. Its electronegative glutamic and aspartic acid residues as well as the putative Ca^{2+} binding motif make it bind tightly to hydroxyapatite. OPN is a major inhibitor of the mineralization process, since binding of OPN to hydroxyapatite inhibit the growth of hydroxyapatite crystals. *(* ***Steitz SA et al 2002, Hunter et al 1996, Pampena et al 2004)***[16,17,18]**.**

Osteoclasts develop from precursor cells in monocyte/macrophage family to become giant multinucleated cells capable of resorbing bone. The maturation and differentiation of macrophages to osteoclasts are dependent on two factors secreted or expressed by stromal cells/ osteoblasts : (i) macrophage colony stimulating factor and

(ii)receptor for activation of NF-kB ligand (RANKL) ***(Teitelbaum 2000)***[19]. Engagement of theses cytokines with their receptors on the osteoclast precursor cells induces maturation of osteoclast. . Osteoprotegerin (OPG) is a decoy receptor for RANKL. It is secreted by osteoblasts / stromal cells and inhibits the interaction of RANKL with RANK and thereby inhibits osteoclastogenesis.

OPN does not seem to be an essential factor in the development of osteoclasts during normal bone development since osteoclast number and distribution are normal in OPN knock-out mice ***(Rittling et al 1998, Liaw et al 1998)***[20,21]. However, several studies show that in pathological conditions, OPN is of importance in osteoclastogenesis. Parathyroid hormone induced RANKL signalling normally results in either an increase in osteoclast number and/or activation , but this increase is disrupted in the absence of OPN ***(Ihara et al 2001)***[22]**.** Neutralization of OPN suppresses osteoclastogenesis , whereas addition of OPN enhances osteoclastogenesis ***(Yamate et al 1997, Ishii et al 2004)***[23,24]**.** OPN stimulate osteoclast migration ***(Faccio et al 1998, 2002, Chellaiah & Hruska 2003)***[25,26,27]**.** Osteoclasts deficient in OPN do not migrate and are unable to resorb bone ***(Chellaiah & Hruska 2003, Tani-Ishii et al 1997)***[27,28]**.**

In mineralized tissue, OPN is secreted by both osteoclasts and osteoblasts (***Yamate et al 1997, Dodds et al 1995, Merry 1993)***[23,29,30]**.** It is an inflammatory mediator whose levels are found to commensurate with the progression of periodontal disease in gingival crevicular fluid and in plasma. The concomitant increase in plasma is caused by spillage or overflow from the diseased periodontal tissues or produced by circulating activated macrophages ***(Sharma CG and Pradeep AR 2007)***[31]**. *Sharma*** reported that osteopontin (OPN) concentrations increased proportionally with the progression of disease and when nonsurgical periodontal treatment was provided, GCF and plasma OPN levels were significantly reduced.

Non-surgical therapy:

The main goal of the treatment of patients with periodontitis is to establish proper infection control, i.e, to reduce the bacterial load below the individual threshold level for disease. Cause-related periodontal therapy is usually the initial phase of periodontal treatment. The aims are to eliminate supra- and sub-gingival bacterial deposits and to prevent their recurrence ***(Haffajee et al. 1997)***[32]**.** Even in the most severe cases of periodontal disease, cause-related periodontal therapy most often precedes surgical therapy. This is done so that the active periodontal infection is reduced and the overall tissue quality is improved prior to surgery. This procedure may also limit the areas requiring surgery.

The aims can be achieved by the following procedures/measures:

- Motivate the patient to understand periodontal disease and the importance of co-operation in order to combat the disease. Optimal treatment results and stable long term conditions depend on optimal self-performed oral hygiene and compliance towards regular maintenance visits.
- Provide the patient with custom-made oral hygiene instruction and reassess the results from time to time and reinforce the technique, if necessary. Maintaining optimal personal hygiene is paramount because at least one study showed that instrumentation alone without improving patient's plaque control may lead to microbial repopulation of the instrumented sites shortly after scaling and root planing ***(Magnusson et al. 1984)***[33]**.**

Non-surgical periodontal therapy include scaling and root planing to remove bacterial plaque and calculus subgingivally by mechanical means, using either manual instruments such as hand scalers/curettes or machine-driven instruments such as sonic or ultrasonic scalers. ***Badersten et al (1981)[34], Torfason et al (1979)[35]*** have shown that both instrumentation approaches could achieve similar results, but according to ***Leon & Vogel (1987)[36]*** ultrasonic instruments might be more suitable in furcation areas.

Although the word "planing" means to remove substance for achieving a smooth surface, ***Nyman et al (1988)[37]*** showed that it was not necessary to plane the root surfaces until smooth or to remove the so called "diseased/ contaminated" cementum**.** A clinical study by ***Oberholzer & Rateitschak 1996[38]*** concluded that the establishment of a smooth and hard root surface was not a critical factor in periodontal therapy**.**

Non-surgical treatment improves clinical parameters including bleeding on probing, probing depths and probing attachment levels, provided that patients can obtain and maintain a proper plaque control **.**

Singletary et al 1982[39], Greenewell et al 1984 [40], Lavanchy et al 1987 [41] reported a significant reduction in gingival inflammation 1-3 months after phase I therapy. ***Hughes & Caffesse 1978 [42]***demonstrated that reduction of probing depth following mechanical instrumentation results from a combination of gain in clinical attachment and increase in gingival recession **.** ***Proye et al 1982 [43]*** noted recession after one week and a gain of clinical attachment by 3 weeks after phase I therapy. After a single episode of scaling and root planing, pockets were reduced to 1.36 mm. This consisted of 0.84 mm recession and 0.52 mm attachment gain.

Morrison et al 1982 [44] in another examination of data from the previously mentioned 8-year longitudinal study analyzed the effect of gingivitis scores on probing depth and attachment levels. For pockets 1 to 3 mm and 4 to 6 mm there was no difference

in pocket reduction maintenance. For attachment there was no difference in 1 to 3 mm probing depths and in 4 to 6 mm pockets, lower gingivitis scores had better gain the first 2 years but thereafter no difference was recorded. For 7 to 12 mm pockets, the lower gingivitis scores seemed to result in better probing levels and attachment gain for the first 3 years but this was not maintained throughout the experiment. The severity of gingivitis did not affect the maintenance of pocket depth reduction or clinical attachment levels.

Ramfjord et al 1982 [45] reported that deepest sites demonstrated the greatest pocket reduction after instrumentation. They conducted a study in pockets with 1-3mm, 4-6mm and ≥7mm for 5years and found the greatest pocket reduction in group with ≥7mm pocket depth. But they also found a greater amount of recession in this group.

Cobb 1996 [46] reported that for shallow pockets with initial probing depth 1-3mm, the mean reduction of probing pocket depth (PPD) was 0.03mm and loss of probing attachment level (PAL) was 0.3mm. For moderate pockets with initial PPD of 4-6mm, the mean reduction of PPD was about 1.29mm and the PAL gain was 0.55mm **.** For deep pockets with initial PPD ≥ 7mm, the mean reduction of PPD was about 2.16mm and the PAL gain was 1.19mm .

The number of sites that bleed on probing also markedly reduces following nonsurgical therapy. ***Cobb (2002)***[47] reviewed many studies and found that the mean reduction in bleeding on probing from baseline levels was about 45%.

Non-surgical periodontal therapy has been proved to be effective in controlling the disease in most patients ***(Badersten et al. 1984, Claffey & Egelberg 1995)***[48,49]. Furthermore, most of the stabilized periodontal conditions can be maintained throughout time if the patients are committed to have supportive periodontal care ***(Axelsson & Lindhe 1981, Renvert & Persson 2004)***[50,51]**.**

Philstrom et al. (1981)[52], in a 4-year study utilizing multi-rooted teeth, compared scaling and root planing to modified Widman surgery. Seventeen patients received thorough scaling and root planing as well as oral hygiene instructions. A modified Widman flap was then performed on one half of each subject's dentition. Patients were recalled 3 to 4 times a year for 4 years. The data were separated into 3 groups by initial pocket depth; 1 to 3 mm, 4 to 6 mm, and > 7 mm. Both methods resulted in increased probing depth and loss of attachment in the 1 to 3 mm group, in the 4 to 6 mm group both procedures resulted in reduction in probing depth and maintenance of attachment levels with the root planing resulting in slightly more gain in attachment. The > 7 mm group showed the greatest reduction in probing depth and gain in attachment with better results in the flap procedures. The results indicate that both procedures were effective in treating moderate to advanced periodontitis. The additional flap procedure tended to result in greater probing reduction and attachment gain for deeper pockets.

Philstrom et al. (1983)[53] in a second report analyzed the 6.5 year results of the previous study. This report concludes that scaling and root planing alone or in combination with modified Widman flap surgery resulted in sustained decreases in gingivitis, plaque, and calculus and neither procedure appears to be superior with respect to these parameters. Seventeen patients diagnosed with moderate-advanced periodontitis were utilized in a split-mouth design study to compare the effects of scaling and root planing alone and combined with modified Widman flap surgery. Data were collected at baseline, 6 months following active therapy and every year up to 4 years, then at 5 1/2 and 6 1/2 years. Probing depth did not change for 1 to 3 mm pockets treated by either scaling and root planning alone or in combination with modified Widman flap surgery. For pockets 4 to 6 mm, both treatment procedures resulted in equally effective sustained pocket reduction. Deep pockets (> 7 mm) were initially reduced more by the flap procedure. After 2 years,

no consistent difference between treatment methods was found in degree of pocket reduction. For pockets initially 4 to 6 mm in depth, attachment level was maintained by both procedures. Pockets > 7 mm in depth treated by either procedure resulted in a sustained gain in attachment.

Philstrom et al. (1984)[54] in a third report examined the response of molar and non-molar teeth to scaling and root planing alone or scaling and root planing plus a flap procedure. At 6 1/2 years, non-molar teeth had an average of about 1.0 mm less probing depth than molar teeth irrespective of type of procedure performed. There was greater probing depth and more apical attachment level on molar than on non-molar teeth treated by either method for 4 to 6 mm pockets. In > 7 mm pockets, the flap resulted in less pocket depth on non-molars than molars, but no difference in the attachment level for either method. Nineteen of the 453 teeth included in the study were extracted throughout the study; 11 of these were extracted after therapy.

Hill et al. (1981)[55] published a 2-year study of scaling and root planing compared to modified Widman surgery. This 90-patient study included multi-rooted teeth. Following a hygienic phase which included scaling and root planing and oral hygiene instructions;, each quadrant was treated by 1 of 4 treatments (pocket elimination, modified Widman flap (MWF), subgingival curettage, and scaling and root planing). Measurements which included pocket depth and attachment levels were taken at the initial exam, after the hygienic phase and 1 and 2 years after treatment. In the 1 to 3 mm crevices there was a slight loss of attachment after all types of treatments. In the 4 to 6 mm pockets there was a significant reduction in probing depth after all modalities with the greatest reductions after pocket elimination and modified Widman flap, and a loss of attachment for pocket elimination and a gain for curettage and scaling. In the > 7 mm pockets there was a significant reduction after all modalities with the greatest reduction after pocket

elimination, and no significant differences in attachment results among the 4 methods. None of the surgical modalities had any better effect than scaling and root planing alone in maintenance of periodontal support which was not directlyrelated to reduction in pocket depth.

Lindhe et al. (1982A)[56] reported results of a 2-year study of 15 patients comparing scaling and root planing to modified Widman surgery. Mutilated teeth were included in this study. Patients with advanced periodontal disease were entered into a split mouth design to compare the results of subgingival debridement performed in conjunction with a modified Widman flap or scaling and root planning alone. Scaling and root planing took 6 to 8 hours over a 4- week period. Oral hygiene and the gingival condition in both groups improved significantly. Both treatments resulted in a decrease in probing depth. Initial values were 4.2 and 4.1 mm and decreased to 2.4 and 2.5 (surgery) and 2.9 and 2.8 mm (no surgery). Attachment levels improved following non-surgical therapy at 6 and 12 months, but at 24 months returned to baseline values. Surgical treatment resulted in a slight loss of attachment of 0.3 mm. When comparing single-rooted to multi-rooted teeth, there was a trend for slightly better results for single-rooted teeth. These similar results can be maintained over time in patients with proper oral hygiene levels.

Lindhe et al. (1982B)[57] in another report from the previous study determined the critical probing depth for scaling and root planing and modified Widman surgery. Probing depths shallower than the critical probing depth tend to lose attachment following the procedure. The results showed that the critical probing depth for the scaling and root planing group was 2.9 mm ± 0.4 and for the modified Widman's group was 4.2 mm ± 0.2 which indicates that in patients with a large number of shallow probing depths, a non-surgical approach is preferable, while in patients with a large number of pockets > 4.2 mm, surgical treatment may result in more gain of attachment. The results also showed that the

level of oral hygiene established during healing and maintenance is more critical for the resulting probing depths and attachment levels than the mode of treatment used.

Lindhe et al. (1984)[58] reported 5-year results of a continuation of the previous study. The results showed that patients who maintained good oral hygiene had more reduction in probing and a greater gain in attachment than patients who failed to perform good plaque control, indicating that the patients' self-performed plaque control had a decisive influence on the long-term effect of treatment. Sites with initial probing depth exceeding 3 mm responded equally well to non-surgical and surgical treatment.

Isidor et al. (1984)[59]**,** in a 6-month study on single-rooted teeth, compared 3 treatments utilizing a split-mouth design: Scaling and root planing versus modified Widman surgery versus reverse bevel flap. Seventeen patients were treated for advanced periodontitis. One side of both the maxilla and the mandible were treated with modified Widman flap. On the other side, one quadrant was treated with reverse bevel flap surgery without osseous recontouring, and the last quadrant was treated with scaling and root planing alone. Patients were recalled every 2 weeks, and examination was performed at 3 and 6 months after the completion of treatment. At 6 months scaling and root planing resulted in considerable reduction in pocket depth, but more shallow pockets were obtained following surgical treatment. Clinical gain of attachment was obtained following all 3 modalities but scaling and root planing resulted in slightly more gain of attachment than the 2 surgical procedures.

Isidor et al. (1985)[60] reported 1-year results of the previous study. Lateral incisors, canines, and premolars in the maxilla and mandible in 16 patients diagnosed with advanced periodontitis were used for study. Each patient was then treated with reverse bevel flap surgery in 1 quadrant, modified Widman flap surgery in 2 quadrants, with the fourth quadrant treated with scaling and root planing. They were then recalled every second week

for professional tooth cleaning. The plaque index and bleeding on probing were assessed prior to and 3, 6, and 12 months after treatment. Probing depths and clinical attachment levels were assessed prior to and 1 year after treatment. Radiographs were taken using the bisecting angle technique before and 1 year after treatment, and the bone level was expressed as a percentage of the distance from the apex of the tooth to the normal bone level. Angular bony defects corresponding to 15% or more of the distance between the normal level of the bone and the apex of the involved tooth were located. The results of this study indicate that when comparing modified Widman flap surgery, reverse bevel flap surgery, and scaling and root planing for regeneration of alveolar bone, only the modified Widman flap surgery resulted in significant coronal regrowth of bone in angular bony defects.

Isidor and Karring (1986)[61] reported 5-year results of the previous studies. Sixteen patients with advanced periodontitis were subjected to supra- and subgingival scaling and oral hygiene instructions. This was followed by modified Widman flap, reverse bevel flap, or scaling and root planing. Patients were then recalled regularly for the next 5 years Surgical and non-surgical treatment resulted in pocket reduction which was maintained over the 5 years. All methods were effective in halting the progression of periodontitis. No correlation was found between oral hygiene and recurrence of periodontitis, suggesting subgingival scaling at frequent recalls is an important factor in halting the progression of disease.

Becker et al. (1988)[62] reported 1-year results of a study comparing scaling and root planing, modified Widman surgery, and osseous surgery utilizing a split mouth design. The study population consisted of 16 patients with 2 or more sites with > 6 mm of clinical attachment loss in the posterior dentition. All patients had a baseline examination including the plaque index, gingival index, probing depth, clinical attachment levels, mobility, and

furcation status. The probing depths were classified as 1 to 3 mm; 4 to 6 mm; or > 7 mm. The clinical attachment level measurements were classified as 0 to 2 mm; 3 to 5 mm; and > 6 mm. Quadrants were randomly assigned to 1 of 3 treatment groups: scaling and root planing, modified Widman flap surgery, or osseous surgery. At 1 year post-treatment, osseous and modified Widman surgery had significantly greater probing reduction when compared to scaling and root planing. For pockets > 7 mm, osseous and modified Widman surgery had significantly greater reduction when compared to scaling and root planing. For pockets 1 to 3 mm, osseous surgery had significantly greater clinical attachment loss when compared with scaling and root planing. The results indicate that at 1 year, scaling and root planing, osseous surgery, and the modified Widman procedure were equally effective in treating moderate to advanced periodontitis.

Kerry et al. (1990)[63] reported 5-year probing depth results of the previous study. Sixteen patients with moderate periodontitis were treated in private practice by periodontists highly competent in performing scaling and root planing, modified Widman flap, and osseous surgery. Patients were evaluated for 5 years. At the 5-year evaluation, plaque and gingival indices were reduced and maintained throughout the study with no difference between treatment methods; 1 to 3 mm probing depths increased insignificantly but were stable at 3 years; 4 to 6 mm pockets were reduced significantly, but diminished over time. There was a difference between scaling and root planing compared to osseous surgery at 3 and 4 years, but not at 5. Similar trends were found for > 7 mm pockets. All 3 procedures reduced pocket depth significantly, with no difference between procedures at 5 years.

Becker et al. (1990)[64] reported 5-year attachment level and gingival recession results of the previous studies. Sixteen patients were treated for moderate periodontitis

with either scaling and root planing, modified Widman flap, or osseous surgery. Evaluation were made after the hygienic phase, postsurgery, 6 weeks, 6 months, and at yearly intervals for 5 years. Pockets 1 to 3 mm showed significant loss of attachment; 4 to 6 mm pockets, as well as > 7 mm pockets, showed an insignificant gain of clinical attachment with no difference among procedures. All procedures produced significant recession postsurgery. It was concluded that all techniques behave similarly regarding clinical attachment levels and gingival recession.

Kaldahl et al. (1988)[65] reported 2-year results of a split mouth design study of multi-rooted teeth that compared supragingival scaling to subgingival scaling to modified Widman surgery to osseous surgery. Eightytwo patients with moderate to advanced periodontitis had each of 4 quadrants randomly assigned to receive coronal scaling , subgingival scaling and root planning , root planing plus modified Widman flap, flap with osseous resection. Approximately 20% of the scaling teeth were retreated. The osseous resection group showed the greatest reduction in probing depth followed by modified Widman, subgingival scaling and root planing, and coronal scaling. In deep sites modified Widman, subgingival scaling and root planing , and osseous resection demonstrated the largest gain in attachment while coronal scaling was the least.

Kaldahl et al. (1990)[66] reported 2-year results of the previous study that compared the site response. Eighty-two patients with moderate to advanced periodontitis were treated in a split mouth design with coronal scaling , root planing , modified Widman flap , and flap with osseous resection , followed by maintenance treatment for 2 years. Four tooth/site groupings were evaluated: 1) interproximal sites of single- rooted teeth (Tl); 2) facial and lingual sites of single-rooted teeth (T2); 3) nonfurcation sites of molar teeth (T3); and 4) furcation sites of molar teeth (T4). The sites were further subdivided by their initial probing depth severity (1 to 4 mm, 5 to 6 mm, and > 7 mm). The results showed that

single-rooted sites > 5 mm had a greater mean probing depth reduction and greater probing attachment gain than did the molar sites. Furcation sites showed a greater increase in probing depth and loss of attachment during the 2 years of maintenance. No therapy had a distinct advantage over another in enhancing the relative response of a particular tooth/site group to the other groupings.

Kalkwarf et al. (1992)[67] reported 2-year results of the 2 previous studies that analyzed patient preference of treatment method. Seventy-five patients were evaluated using an interview after 3 years of maintenance care. Each quadrant in each subject was randomly assigned to 1 of 4 types of periodontal therapy: 1) coronal scaling; 2) coronal scaling plus subgingival scaling and root planing; 3) coronal scaling / scaling and root planning followed by modified Widman surgery ; or 4) Coronal scaling/ scaling and root plaing followed by flap with osseous resectional surgery. During the hygienic phase of therapy, patients were instructed in plaque control and teeth were instrumented with sealers and currettes. Maintenance therapy was performed at 3-month intervals by a dental hygienist. At the conclusion of 3 years of maintenance care, a 7 question interview was conducted with each patient to obtain perceptions regarding the results of therapy in each region of their mouth. The results of this study indicate that the ability of the patient to cope with post-therapy sequelae following either coronal scaling, root planing, modified Widman surgery, or flap with osseous resectional surgery is not significantly different.

Cercek et al. (1983) [68] reported results of a 2-year study that compared supragingival plaque control to subgingival plaque control to scaling and root planing. Seven patients with chronic periodontitis were monitored during 3 phases of treatment: 1) tooth brushing and flossing; 2) Perio-Aid used subgingivally; and 3) subgingival debridement. Plaque scores ranged from 38 to 99% with a mean of 74% at the initial exam. These scores were reduced to 5 to 15% and were maintained throughout the study. The

mean bleeding score of 71.7% was reduced to 40.9% in Phase I, no change in Phase II, and reduced to 23% in Phase III. Deeper sites showed more bleeding than shallower sites throughout the study. The mean probing depth of 4.4 mm was reduced to 4.0 mm in Phase I, no improvement in Phase II, and reduced to 3.2 mm after instrumentation. Probing attachment level showed a slight loss through Phase II, but improved attachment levels were found after instrumentation. An increasing gingival recession was noted during the study. Minimal effect was derived from patient-performed plaque control, whether supra- or subgingival. The bulk of the effect was derived from professional subgingival instrumentation (SRP).

Badersten et al. (1981)[34] in a 13-month study of patients with moderate periodontitis compared the effect of hand versus ultrasonic instrumentation on attachment levels of single-rooted teeth. Incisors, canines, and premolars in 15 patients with moderately advanced periodontitis were treated by hand and ultrasonic non-surgical therapy. Improvements in plaque scores, bleeding on probing, decreased probing and attachment levels were similar for both treatment methods. It was shown that shallower sites had a slight loss of attachment while deeper sites showed some improvement.

Badersten et al. (1984A)[48] reported 24-month results of a study comparing hand to ultrasonic instrumentation in patients with severe periodontitis. Sixteen patients with severely advanced periodontal disease were treated by hand or ultrasonic non-surgical therapy. Comparable results were obtained by both methods. It was shown that the deep probing depths could be successfully treated non-surgically based on probing depth, probing attachment levels, bleeding on probing, plaque, and gingival recession. It was shown that shallower sites were at risk of losing attachment, while the deep sites were more likely to gain attachment. Deeper residual probing sites were more likely to bleed on probing.

Badersten et al. (1984B)[69] compared the effect of a single session of scaling and root planing to repeated sessions of scaling and root planing. Incisors, canines, and premolars were studied in 13 patients with severe periodontitis. Teeth were instrumented using ultrasonic instruments, and repeated instrumentation in one side of the jaw was performed after 3 and 6 months. A gradual and marked improvement took place during the first 9 months. No differences in results could be observed when comparing the effects of a single versus repeated instrumentation. Deep periodontal pockets in incisors, canines, and premolars may be treated by plaque control and one episode of instrumentation.

Badersten et al. (1985A)[70] reported a study of the effect of operator variability on the results of the scaling and root planing procedure. Twenty patients whose dentition displayed generalized severe periodontal destruction were selected for the study. The incisors, canines, and premolars in either the maxilla or the mandible were studied. The periodontal pockets were debrided using either hand and/or ultrasonic instruments under local anesthesia by a periodontist or by 1 of 5 dental hygienists. A split mouth design was used with measurements of dental plaque, bleeding on probing, probing depth, and probing AL recorded at the initial exam and at every third month by an examiner not involved with treatment. The results indicate that deep periodontal pockets in incisors, canines, and premolars may be successfully treated by plaque control and one episode of instrumentation and that operator variability between highly skilled clinicians is minimal.

Badersten et al. (1985B)[71] examined patterns of probing attachment loss following scaling and root planing. Incisors, canines, and premolars in 33 patients with generalized periodontal destruction were studied for patterns of probing attachment loss. Patients received supra- and subgingival debridement after oral hygiene instructions, and were followed for 24 months. Measurements were made every third month and 7 patterns of probing attachment were identified. Seventy-three percent (73%) of sites showed a gradual

change. Seventeen percent (17) showed an early loss followed by a stabilization of attachment levels. Shallower sites showed a pattern of early loss followed by stabilization while deeper sites showed a gradual loss.

In general, clinicians should assess healing four to six weeks after performing root planning ***(AAP 1989)[72]. Cercek and coworkers (1983)[68]*** noted clinical improvements continued for 8 months, however, most of the healing occurred during the first month.

Similarly, ***Kaldahl and colleagues (1988)[65]*** demonstrated that the repair process extended for 1 year. It appears that the greatest changes with respect to probing depth reduction and gain of clinical attachment can be recorded after 4 to 6 weeks, but gradual repair and maturation of the periodontium may occur over 9 to 12 months.

Longitudinal studies by ***Ramfjord et al (1982)[45]*** indicated that scaling and root planing arrested attachment loss as well as surgical therapy regardless of probing depths. However, information relating to length of therapy, skill level of therapists, compliance of patients with recall intervals and plaque control, responsibilities of clinicians for long-term maintenance and disease activity status need further clarification. Concerns regarding these issues must be integrated into therapeutic decisions, because they can dramatically affect interpretation and application of reported data.

Homeopathic medicine :

Non-surgical therapy remains the corner stone of periodontal treatment. But complementary and alternative therapies have gained popularity among patients in the recent years. Complementary medicine is based on the perspective that everything, including our bodies, is balanced and any disturbance—be it mental, emotional or physical—can cause a diseased state. One such system of alternative medicine is homeopathy.

Homeopathy is a system of alternative medicine originated in 1796 by *Samuel Hahnemann,* based on his doctrine of *similia similibus curentur* ("like cures like"), according to which a substance that causes the symptoms of a disease in healthy people will cure similar symptoms in sick people ***(Lessell 1995)[4].***

Hahnemann believed that the underlying causes of disease were phenomena that he termed *miasms,* and that homeopathic remedies addressed these. The remedies are prepared by repeatedly diluting a chosen substance in alcohol or distilled water, followed by forceful striking on an elastic body, called *succession* ***(Lessell 1995, Dana Ullman 1991) [4,73] .*** Each dilution followed by succussion is said to increase the *remedy's potency*. Dilution usually continues well past the point where none of the original substance remains. Homeopaths select remedies by consulting reference books known as *repertories,* considering the totality of the patient's symptoms as well as the patient's personal traits, physical and psychological state, and life history ***(Dana Ullman 1991)[73].***

Conventional therapy believes that symptoms of an illness are a direct result of that illness and tries to suppress them with the medicine whereas homeopathy sees symptoms as the body's attempt to overcome the illness and seeks to support this process with the remedy, and not to suppress it. Homeopathic remedies are based on a holistic approach to health that addresses illness by stimulating the body's own healing powers. The idea behind homeopathic medicine is similar to that of getting a vaccination.

The scope for homeopathy in dental practice is wide ranging. Homeopathy is used as an effective adjunct to dental surgery to help alleviate associated pain, bleeding and inflammation. At a more advanced level homeopathy can be used in dental practice to:

- Prevent or inhibit the development of morbid processes in the oral cavity
- Provide adjunctive treatments for a number of defined oral pathologies
- Prevent, limit or ameliorate complications and sequelae of surgical intervention

- Ameliorate dental and soft tissue pain
- Assuage dental phobias and anxieties
- Improve patient tolerance of medications
- Facilitate recovery from dental trauma, restorative treatments and anaesthetic agents.

Constitutional homeopathic prescribing involves analyzing a person's body type, temperament, disposition and behavioural tendencies. Pathological prescribing is a treatment specifically for the disease or ailment. Homeopathy in dentistry offers a combination of these. It is possible to prescribe one medicine to suit the general temperament or psychological state of a patient and another for the particular problem the patient is experiencing. This is of particular value when using homeopathic medicines as an adjunct in the treatment of complex chronic dental pathologies, such as periodontal disease, TMJ dysfunction or Lichen planus.

There are homeopathic medicines for every facet of dental prescribing, and some are specific to certain oral conditions. Homeopathy is not a replacement for the skills used in everyday dental procedures but is a useful complement, particularly in helping apprehensive patients to make dental treatment more acceptable and comfortable. They are safe, nontoxic and non-addictive and produce no known side-effects or interaction with conventional medicines. Consequently, homeopathic medicines can be taken with conventional drugs, by children and during pregnancy. Homeopathic medicines come in different forms including tablets, powders, pills, drops, liquids, tinctures, granules and creams. They are generally administered in one of two ways. Systemic administration involves dissolving the medicine either on or under the tongue from where it is rapidly absorbed. The dose is normally one or two tablets. The tablets should never be handled, as the medicine is impregnated on the surface of the tablets. Alternatively, the tablets can be

tipped from the cap of their container directly into the mouth and sucked or chewed. ***(Peter Darby, 2011)[74].*** At present there is no complete explanation of how homeopathic medicines work, but scientific interest in ultra-molecular dilutions is increasing and there is a growing body of evidence suggesting ultra-molecular solutions can exert biological effects ***(Linde et al, 1994; Belon et al, 2004; Witt et al, 2007)[75,76,77].***

Calcarea fluorica is a homeopathic medicine with chemical union of lime and fluoric acid which gives a remedy with a new nature and properties. However conversant one may be with either or both of these elements, the curative powers held in this double remedy cannot be predicted. It is used in case of unnatural looseness of teeth, with or without pain; teeth becoming loose in the sockets and in case of toothache if any food touches the tooth. ***(W Boericke, 2000)[78].*** This medicine has been used previously in dental field to check the remineralization efficacy on artificial carious lesions on enamel (***Bansal et al, 2014)[79]***. But the present study is the only study till date to access the effectiveness of calc-f tablets on periodontium.

Materials and Methods

MATERIALS AND METHODS:

Source:

The study population needs to be selected from the outpatient sections of the Department of Periodontitis, TNGDC and Hospital, Chennai.

SAMPLE SIZE : 20

INCLUSION CRITERIA:

- Patients with age group between 30-55 years.
- Systemically healthy patients with clinical attachment level (CAL) ≥ 3mm in proximal sites of two non-adjacent teeth; bone loss confirmed by periapical radiographs; bleeding on probing (BOP) and probing depth (PD) 4-6mm.
- Patients who have not undergone any type of regenerative periodontal therapy over a period of six months prior to the initial examination.
- Patients without any antibiotic treatment in last three months.
- Patients with established willingness and ability to perform adequate oral hygiene.

EXCLUSION CRITERIA:

- Patients who are suffering from any known systemic diseases or immune-compromised
- Patients who had received any surgical or non-surgical therapy six months prior to the start of the study
- Patients who had received any antibiotic therapy in the last three months
- Tobacco users and alcoholics were excluded
- Pregnant and lactating females were not included in the study

SUBJECTS:

- A total of 20 patients suffering from chronic periodontitis were divided into 2 groups based on intervention with 10 patients in each group. Age group 30-55 years.
- Group I: SRP only
- Group II: SRP + homeopathy drug (*Calcarea fluorica 30c)*

STUDY DESIGN:

The study is of randomized controlled trial type. The study participants will be recruited prospectively in this study.

Sex: Either sex

Ethical clearances were obtained from the Institutional Ethical Committee and ethical principles were meticulously followed throughout the study. After explaining the study protocol , informed consent was obtained from all the selected subjects . A thorough medical and dental history of the subjects was taken. All the subjects underwent full-mouth periodontal probing and charting and clinical and laboratory evaluation.

METHOD OF COLLECTING DATA:

Armamentarium:

For Clinical Examination:

- Mouth mirror
- Williams periodontal probe
- Kidney tray

- Cotton roll
- Sterilized disposable gloves, head cap, facemask

For Phase I therapy:

- Mouth mirror
- Williams probe
- Kidney tray
- Cotton rolls
- Sterilized disposable gloves, head cap, facemask
- Disposable syringes
- Local anaesthetic solution (Lignocaine)
- Hu-Friedy Gracey Currettes

For collection of blood sample

- Sterile cotton.
- Surgical spirit
- Disposable syringe with 20 gauge needle.
- Tourniquet.

CLINICAL PARAMETERS ASSESSMENT

- The following clinical parameters were evaluated for all the subjects:

1. Plaque index – ***Silness and Loe 1964***[80]

2. Gingival bleeding index – ***Ainamo and Bay 1975*** [81]

3. Probing depth in mm (PD) – ***Carranza 10th ed*** [82]

4. Clinical attachment level in mm (CAL) – ***Carranza 10th ed***[82]

***Plaque Index (Silness and Loe 1964*)**

All teeth were examined at 4 sites each (disto-facial, facial, mesio-facial, lingual / palatal) and were scored as follows:

Scoring Criteria:

Score 0: No plaque in the gingival area.

Score 1: A film of plaque adhering to the free gingival margin and adjacent area of the tooth. The plaque is recognized only by running a probe across the tooth surface.

Score 2: Moderate accumulation of plaque within the gingival pocket and on the gingival margin and / or adjacent tooth surface that can be seen by the naked eye.

Score 3: Abundance of soft deposits within the gingival pocket and / or on the gingival margin and adjacent tooth surface.

Calculation:

Plaque index per tooth = Total score/4

Plaque index per individual = Total P I per tooth / Total number of teeth examined

Interpretation:

Score 0 – Excellent oral hygiene

0.1 to 0.9 – Good oral hygiene

1.0 to 1.9 – Fair oral hygiene

2.0 to 3.0 - Poor oral hygiene

Gingival Bleeding Index *(Ainamo & Bay 1975)*

Starting distobuccally, the probe was gently inserted into the sulcus and run to the buccal and mesial surfaces of every tooth at an angle of about 45º. This was repeated for all the teeth present. Similarly probing was carried out at palatal/lingual sites. The total

number of bleeding sites per tooth was thus recorded for every tooth except the third molar.

Scoring Criteria:

Positive score (1) - Presence of bleeding within 10 seconds

Negative score (0) - Absence of bleeding

$$\% \text{ of bleeding sites} = \frac{\text{Total number of positive score} \times 100}{\text{Total number of surfaces of all teeth}}$$

Probing Pocket Depth (PPD)

Probing Pocket Depths were measured from the gingival margin to the base of the pocket in millimeters using William's Periodontal Probe. The probe was walked within the gingival sulcus along the circumference of the tooth. Keeping the probe parallel to the long axis of the selected tooth, six measurements were made per tooth (Mesiobuccal, Distobuccal, Midbuccal, Mesiolingual, Distolingual and Midlingual).

Clinical Attachment Level (CAL)

Clinical Attachment Level was measured from the Cemento – Enamel Junction (CEJ) to the base of the pocket using William's Periodontal Probe.

When the gingival margin was located on the anatomic crown, the level of the attachment was determined by subtracting from the probing depth, the distance from the gingival margin to the CEJ. If both were the same, the loss of attachment was calculated to be zero.

When the gingival margin coincided with the CEJ, the loss of attachment was calculated as equaling the probing depth.

When the gingival margin was located apical to the CEJ, the loss of attachment was greater than the probing depth and therefore the distance between the CEJ and the gingival margin were added to the PD.

Three measurements were made on the buccal aspect and three on the lingual aspect of each tooth – total of six sites per tooth (Mesiobuccal, Midbuccal, Distobuccal, Mesiolingual, Midlingual, and Distolingual).

ESTIMATION OF OSTEOPONTIN

METHOD OF COLLECTION OF BLOOD SAMPLE

Venous blood was drawn from the participants selected for the study. The subjects were informed, and consent was taken. They were made to tighten a fist so that vein was more palpable, and antecubital vein was selected for venipuncture. A tourniquet was applied about 1-2 inches above the antecubital fossa. After cleansing the puncture site with 10% isopropanol solution, blood was withdrawn using a syringe with 24 gauge needle. Tourniquet was released as the blood flow began. After drawing 3 ml of blood, sterile cotton ball was placed on the puncture site and needle was withdrawn. The subjects were instructed to apply mild finger pressure on the site for few minutes to avoid oozing out of blood.

Quantitative determination of osteopontin in patient's plasma was done by enzyme-linked immunosorbent assay (ELISA) method.

PRINCIPLE OF THE ASSAY

The kit uses a double-antibody sandwich enzyme-linked immunosorbent assay (ELISA) to assay the level of human osteopontin (OPN) in samples. Osteopontin (OPN) was added to monoclonal antibody enzyme well which was pre-coated with human osteopontin monoclonal antibody. This was incubated and then osteopontin antibodies labeled with biotin was added and combined with Streptavidin-HRP to form immune complex. Incubation was carried out and washed again to remove the uncombined enzyme. To this chromogen solution A, B was added which resulted in change of color to blue and at the effect of acid, the color changed to yellow. The chroma of color and the concenthumanion of the human plasma osteopontin sample were positively correlated.

OSTEOPONTIN DETERMINATION

PROCEDURE

1. All reagents were brought to room temperature before use.
2. Standard no.5 (48ng/ml) in duplicate

   Standard no.4 (24ng/ml) in duplicate

   Standard no.3 (12ng/ml) in duplicate

   Standard no.2 (6ng/ml) in duplicate

   Standard no.1 (3ng/ml) in duplicate , were pipette out.
3. Inject samples:① Blank well: no samples or OPN-antibody labeled with biotin, Streptavidin-HRP were added, only Chromogen solution A and B, and stop solution were allowed; other operations were the same.② Standard wells: standard 50µl, Streptavidin-HRP 50µl (since the standard already has combined biotin antibody, it was not necessary to add the antibody) were added;③ To be test wells:

sample 40µl, was added and then both OPN-antibody 10µl and Streptavidin-HRP 50µl were added. Then sealing memberance was sealed, gently shaken and incubated for 60 minutes at 37 ℃.

4. Confection: 30×washing concentrate was diluted 30 times with distilled water as standby.
5. Washing: The memberance was removed carefully, the liquid drained and the remaining water was shook away.
6. Chromogen solution A 50µl, then chromogen solution B 50µl were added to each well, gently mixed and incubated for 10 min at 37℃ away from light.
7. Stop: Stop Solution 50µl was added into each well to stop the reaction (the blue changes into yellow immediately).
8. Final measurement: after taking blank well as zero, the optical density (OD) was measured under 450 nm wavelength which was carried out within 15min after adding the stop solution.
9. According to standards' concentration and the corresponding optical density values, the standard curve linear regression equation was calculated. The optical density values of the sample were applied on the regression equation and the corresponding sample's concentrations were calculated.

SUMMARY

Patient blood sample collected

Sample added to microtitre wells

Preparing reagents and standards

Add prepared samples and standards, antibodies labeled with enzyme, reacting 60 minutes at 37 ℃

Plate washed five times, adding Chromogen solution A, B, reacting 10 minutes at 37 ℃

Add stop solution measure the optical density value within 10min

Calculation

DRUG USED IN THE STUDY

Calcarea fluorica (30c) :

Calcarea fluorica, also called calcium fluoride, fluoride of lime, calcii fluoridum or fluorspar, is a naturally occurring mineral. Calcium fluoride is an important mineral for teeth and bones and is also found in the tissue fibers that make up skin, vessel walls and connective tissue. It prevents unnatural looseness of teeth, with or without pain thus prevent tooth loosening in the socket. Also used for gum-boil, with hard swelling on the jaw, cracked appearance of the tongue, with or without pain, induration of tongue, hardening after inflammation and for toothache with pain if any tooth touches the tooth. It is a powerful tissue remedy for hard

stony glands, varicose and enlarged veins and malnutrition of bones. Also used for hard swellings on the cheek with pain or toothache and hard swellings on jaw bone. It is useful for building the health and strength of teeth and bones during childhood, and after a serious injury to the bones, especially a fracture. It is also helpful for lumps that develop on bones (after a bad bruise to a bone or a fracture) or around joints (after a bad sprain).

***Photograph 1*: Calcarea Fluor**

Photograph 2 **: Group I Patient Before Therapy**

Photograph 3 **: Group I Patient 3 Months After Therapy**

Photograph 4 **: Group II Patient Before Therapy**

Photograph 5 **: Group II Patient 3 Months After Therapy**

Photograph 6 **: Armamentarium for blood collection**

Photograph 7 : **Collection of venous blood sample**

Photograph 8 **: Armamentarium for phase I therapy**

Photograph 9 **: ELISA Reader**

Statistical Analysis

STATISTICAL ANALYSIS

In the present interventional study, 20 subjects with generalized chronic periodontitis were included, 10 among them were categorized as **Group I** and the other 10 were categorized as **Group II.** There were 5 females and 5 males in each group, with a mean average of 39.40± 6.89 years in Group I, 38.06 ± 8.25years in Group II. The parameters assessed were plaque index, gingival bleeding index, probing depth, clinical attachment level (clinical) and serum osteopontin level (laboratory) at baseline and 3 months post-operatively.

The statistical analysis was done using the computer software program SPSS version 16.0 (Statistical Package for Social Science, Version 16).

Statistical Tests used:

1. Independent sample test – For inter group analysis
2. Chi-Square test - For gingival bleeding index
3. Paired Sample test – For intragroup analysis
4. Pearsons Correlation test – For correlation

Results

RESULTS

1. PLAQUE INDEX

Intragroup comparison

Group I: The mean plaque index score at baseline was 2.42± 0.23 and at 3 months was 0.99 ±0 .27. The mean difference in plaque score from baseline to 3 month was statistically significant (p=0.000).

Group II: The mean plaque index score at baseline was 2.45 ± 0.31 and at 3 months was 1.04 ± 0.32. The mean reduction in plaque score from baseline to 3 months was statistically significant (p=0.000).

Intergroup comparison

Mean difference between group I and group II at baseline was -0.027 which was statistically not significant (p=0.831) and at 3 months post-op was -0.055 which was also statistically insignificant (p=0.685).

2. PROBING POCKET DEPTH

Intragroup comparison

Group I: The mean PPD at baseline was 3.66 ±1.06 and at 3 months was 2.64 ±0.76. The mean reduction in PPD from baseline to 3 months was statistically significant (p=0.003).

Group II: The mean PPD at baseline was 3.84±0.62 and at 3 months was 2.09±0.72. The mean reduction in PPD from baseline to 3 months was statistically significant (p=0.000).

Intergroup comparison

Mean difference between group I and group II at baseline was -0.18 which was statistically non-significant (p=0.649) and at 3 months post-op was 0.55 which was statistically non-significant (p=0.115).

3. CLINICAL ATTACHMENT LEVEL

Intragroup comparison

Group I: The mean CAL at baseline was 3.97 ±1.10 and at 3 months was 3.01±0.97. The mean reduction in CAL from baseline to 3 months was statistically significant (p=0.006).

Group II: The mean CAL at baseline was 4.25 ±0.72 and at 3 months was 2.89±0.65. The mean reduction in CAL from baseline to 3 months was statistically significant (p=0.000).

Intergroup comparison

Mean difference between group I and group II at baseline was -0.28 which was statistically non-significant (p=0.506) and at 3 months post-op was 0.116 which was statistically non-significant (p=0.757).

4.OSTEOPONTIN

Intragroup comparison

Group I: The mean osteopontin at baseline was 1.263±9.97 and at 3 months was 1.047±6.35. The mean reduction in serum osteopontin from baseline to 3 months was statistically significant (p=0.000).

Group II: The mean serum osteopontin at baseline was 1.261 ±8.69 and at 3 months was 1.046±8.27. The mean reduction in serum osteopontin from baseline to 3 months was statistically significant (p=0.000).

Intergroup comparison

Mean difference between group I and group II at baseline was 1.60 which was statistically not significant (p=0.707) and at 3 months post-op was 0.500 which was statistically non-significant (p=0.881).

TABLE 1 : MASTER CHART – GROUP 1 (*CLINICAL PARAMETERS)*										
S. NO	**AGE**	**SEX**	**BASELINE PI**	**BASELINE PPD**	**BASELINE CAL**	**BASELINE GBI**	**3MONTH PI**	**3MONTH PPD**	**3MONTH CAL**	**3MONTH GBI**
1.	45	F	2.56	5.13	5.78	1	0.67	3.34	3.65	0
2.	42	M	2.53	4.17	4.21	1	0.57	2.17	2.56	1
3.	31	M	2.23	3.88	4.60	1	1.08	2.03	2.12	0
4.	39	M	2.30	4.51	4.60	1	1.01	3.26	4.33	0
5.	40	M	2.57	3.68	4.11	1	1.24	3.13	3.86	0
6.	42	F	2.34	4.26	4.36	1	1.21	2.83	3.39	0
7.	34	F	2.46	2.42	3.01	1	0.68	2.90	2.98	0
8.	29	M	1.98	1.46	1.61	1	1.05	0.85	0.90	0
9.	44	F	2.86	3.18	3.48	1	1.42	2.80	3.08	0
10	32	F	2.40	3.96	4.01	1	1.01	3.11	3.18	0

TABLE 2: MASTER CHART – GROUP II (*CLINICAL PARAMETERS)*										
S. NO	**AGE**	**SEX**	**BASELINE PI**	**BASELINE PPD**	**BASELINE CAL**	**BASELINE GBI**	**3MONTH PI**	**3MONTH PPD**	**3MONTH CAL**	**3MONTH GBI**
1.	28	F	2.44	3.92	4.25	1	0.55	1.23	3.52	0
2.	44	M	2.33	2.89	3.18	1	0.57	1.18	2.28	1
3.	31	M	2.30	5.01	5.65	1	1.10	3.54	3.88	0
4.	34	F	2.33	4.20	4.90	1	1.05	2.21	3.08	0
5.	32	F	2.87	3.56	3.99	1	1.44	1.38	2.25	0
6.	30	M	2.45	3.28	3.66	1	1.36	1.88	2.03	0
7.	32	F	2.85	3.89	4.04	1	1.44	2.08	2.53	0
8.	38	M	1.66	4.42	4.67	1	0.90	2.57	2.95	1
9.	45	M	2.50	3.27	3.61	1	1.10	2.29	2.68	0
10.	38	F	1.77	4.02	4.55	1	0.97	2.55	3.69	0

TABLE 3 : MASTER CHART – GROUP I (LABORATORY PARAMETER)

S. NO	AGE	SEX	BASELINE OSTEOPONTIN	3 MONTH POST-OP OSTEOPONTIN
1.	45	F	1250	1038
2.	42	M	1274	1052
3.	31	M	1254	1039
4.	39	M	1280	1058
5.	40	M	1265	1048
6.	42	F	1253	1042
7.	34	F	1255	1044
8.	29	M	1267	1050
9.	44	F	1265	1049
10.	32	F	1270	1052

TABLE 4 : MASTER CHART – GROUP II (LABORATORY PARAMETER)

S. NO	AGE	SEX	BASELINE OSTEOPONTIN	3 MONTH POST-OP OSTEOPONTIN
1.	28	F	1252	1036
2.	44	M	1273	1057
3.	31	M	1258	1046
4.	34	F	1275	1058
5.	32	F	1268	1049
6.	30	M	1269	1056
7.	32	F	1255	1042
8.	38	M	1252	1035
9.	45	M	1258	1045
10.	38	F	1257	1043

TABLE 5 : COMPARISON OF PLAQUE SCORES

GROUPS	BASELINE	3 MONTHS POST-OP	P VALUE
GROUP I	2.42 ± 0.235	0.993 ± 0.275	0.000
GROUP II	2.45 ± 0.315	1.048 ± 0.318	0.000

TABLE 6 : COMPARISON OF GINGIVAL BLEEDING INDEX

GROUPS	BASELINE		3 MONTH POST-OP		P VALUE
	ABSENCE	PRESENCE	ABSENCE	PRESENCE	
GROUP I	0	10	9	1	0.000
GROUP II	0	10	8	2	0.001

TABLE 7 : COMPARISON OF PROBING POCKET DEPTH

GROUPS	BASELINE	3 MONTH POST-OP	P VALUE
GROUP I	3.665 ± 1.067	2.64 ± 0.764	0.003
GROUP II	3.846 ± 0.623	2.091 ± 0.723	0.000

TABLE 8 : COMPARISON OF CLINICAL ATTACHMENT LEVEL

GROUPS	BASELINE	3 MONTH POST-OP	P VALUE
GROUP I	3.967 ± 1.103	3.005 ± 0.971	0.006
GROUP II	4.250 ± 0.719	2.889 ± 0.645	0.000

TABLE 9 : COMPARISON OF OSTEOPONTIN LEVEL

GROUPS	BASELINE	3 MONTH POST-OP	P VALUE
GROUP I	1.263 ± 9.978	1.047 ± 6.356	0.000
GROUP II	1.261 ± 8.692	1.046 ± 2.616	0.000

INTERGROUP ANALYSIS USING INDEPENDENT SAMPLES TEST

TABLE 10 : PLAQUE SCORE

Dependent Variable	Mean difference	Std. Error	Sig.	95% confidence level	
				Lower Bound	Upper Bound
Baseline Plaque score	-0.2700	0.124	0.831	-0.288	0.234
3 month Plaque score	-0.055	0.133	0.685	-0.334	0.224
Difference Plaque score	0.028	0.151	0.856	-0.290	0.346

TABLE 11 : GINGIVAL BLEEDING INDEX

Dependent Variable	Mean difference	Std. Error	Sig.	95% confidence level	
				Lower Bound	Upper Bound
Baseline Gingival bleeding index	-0.055	0.126	0.668	-0.319	0.209
3 month Gingival bleeding index	-0.109	0.073	0.153	-0.262	0.044
Difference Gingival bleeding Index	0.054	0.884	0.549	-0.131	0.239

TABLE 12 : PROBING POCKET DEPTH

Dependent Variable	Mean difference	Std. Error	Sig.	95% confidence level	
				Lower Bound	Upper Bound
Baseline PPD	-0.181	0.390	0.649	-1.002	0.640
3 month PPD	0.551	0.332	0.115	-0.148	1.250
Difference PPD	-0.732	0.289	0.021	-1.340	-0.123

TABLE 13 : CLINICAL ATTACHMENT LEVEL

Dependent Variable	Mean difference	Std. Error	Sig.	95% confidence level	
				Lower Bound	Upper Bound
Baseline CAL	-0.283	0.416	0.506	-1.158	0.592
3 month CAL	0.116	0.368	0.757	-0.659	0.891
Difference CAL	-0.399	0.301	0.203	-1.032	0.234

TABLE 14 : OSTEOPONTIN

Dependent Variable	Mean difference	Std. Error	Sig.	95% confidence level	
				Lower Bound	Upper Bound
Baseline Ostepontin	1.600	4.184	0.707	-7.192	10.392
3 month Ostepontin	0.500	3.299	0.881	-6.431	7.431
Difference in Osteopontin	1.100	1.464	0.462	-1.975	4.175

TABLE 15 : GROUP I CORRELATION

Correlations

		PI (3mnths)	GBI (3mnths)	PPD (3mnths)	CAL (3mnths)	OPN (3mnths)
PI3mnths	Pearson Correlation	1	.143	-.010	.059	.092
	Sig. (2-tailed)		.694	.978	.872	.801
	N	10	10	10	10	10
GBI3mnths	Pearson Correlation	.143	1	.428	.368	.025
	Sig. (2-tailed)	.694		.217	.295	.945
	N	10	10	10	10	10
PPD3mnths	Pearson Correlation	-.010	.428	1	.952**	-.020
	Sig. (2-tailed)	.978	.217		.000	.955
	N	10	10	10	10	10
CAL3mnths	Pearson Correlation	.059	.368	**.952****	1	.132
	Sig. (2-tailed)	.872	.295	**.000**		.716
	N	10	10	10	10	10
OPN3mnths	Pearson Correlation	.092	.025	-.020	.132	1
	Sig. (2-tailed)	.801	.945	.955	.716	
	N	10	10	10	10	10

**. Correlation is significant at the 0.01 level (2-tailed).

TABLE 16 : GROUP II CORRELATION

Correlations

		PI (3mnths)	GBI (3mnths)	PPD (3mnths)	CAL (3mnths)	OPN (3mnths)
PI3mnths	Pearson Correlation	1	.146	.238	-.385	.180
	Sig. (2-tailed)		.688	.508	.272	.618
	N	10	10	10	10	10
GBI3mnths	Pearson Correlation	.146	1	.265	-.009	.154
	Sig. (2-tailed)	.688		.460	.980	.671
	N	10	10	10	10	10
PPD3mnths	Pearson Correlation	.238	.265	1	.586	-.205
	Sig. (2-tailed)	.508	.460		.075	.571
	N	10	10	10	10	10
CAL3mnths	Pearson Correlation	-.385	-.009	.586	1	-.471
	Sig. (2-tailed)	.272	.980	.075		.169
	N	10	10	10	10	10
OPN3mnths	Pearson Correlation	.180	.154	-.205	-.471	1
	Sig. (2-tailed)	.618	.671	.571	.169	
	N	10	10	10	10	10

Figure 1 : **Comparison of plaque index between group I and group II**

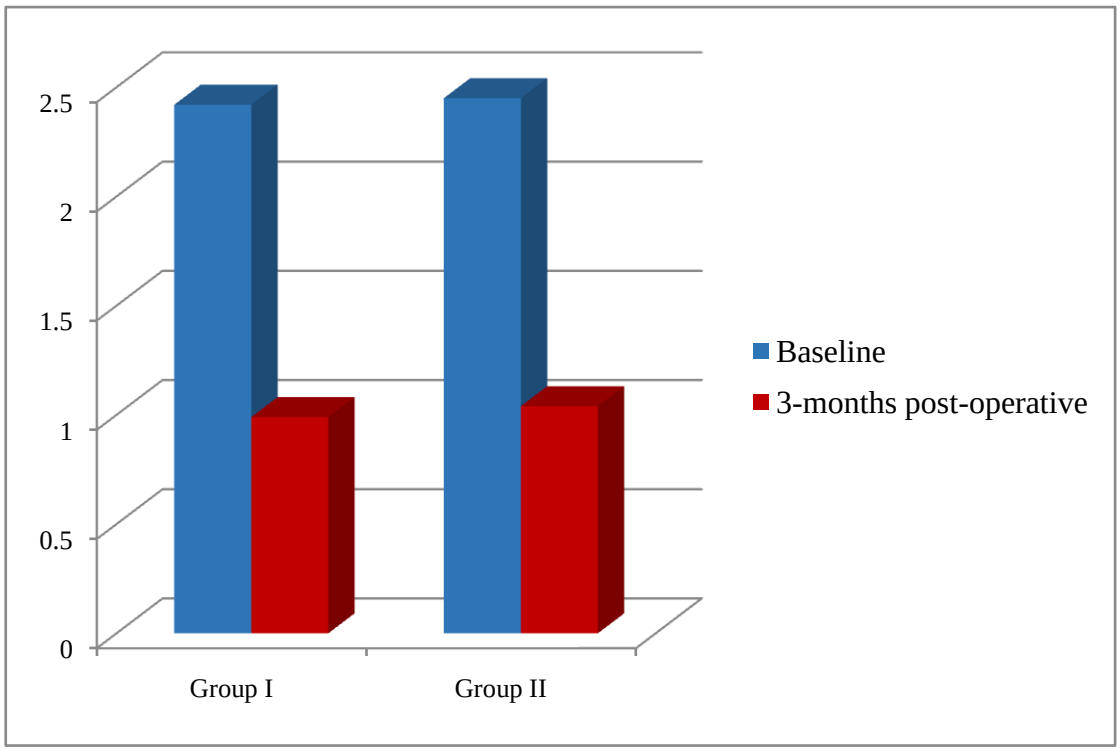


Figure 2 **: Comparison of gingival bleeding index between group I and group II**

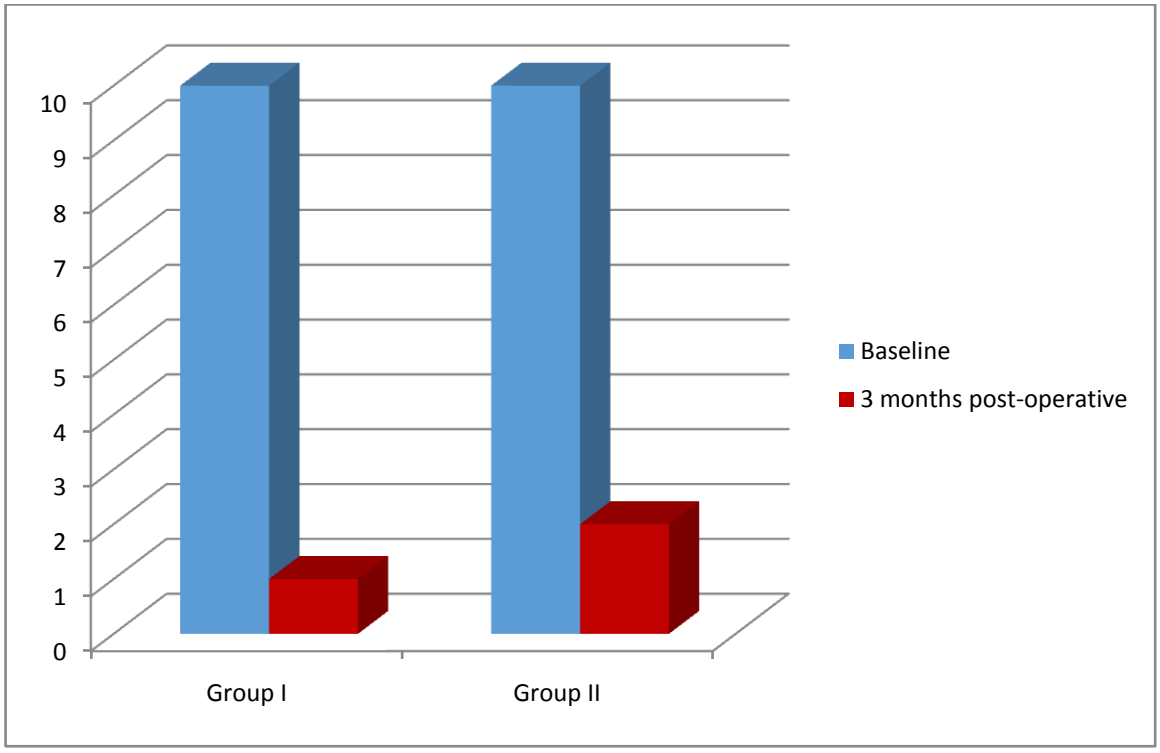


Figure 3 : **Comparison of Probing pocket depth between group I and group II**

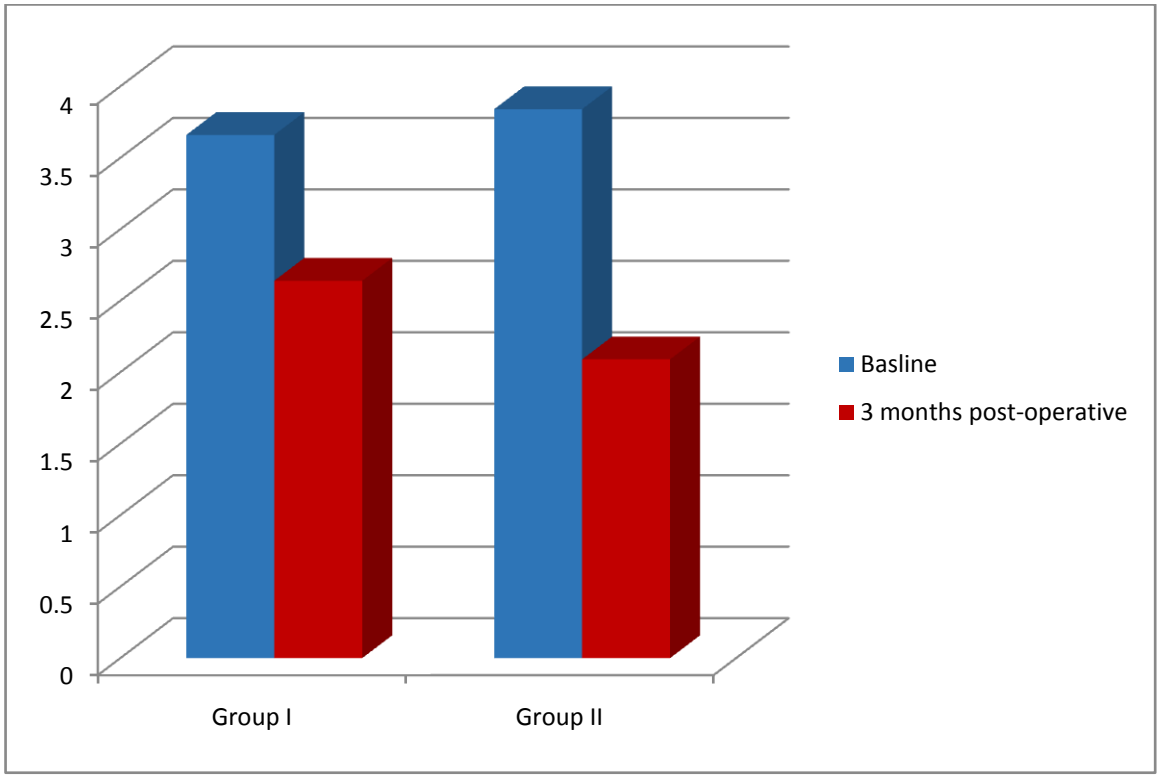


Figure 4 **: Comparison of Clinical attachment level between group I and group II**

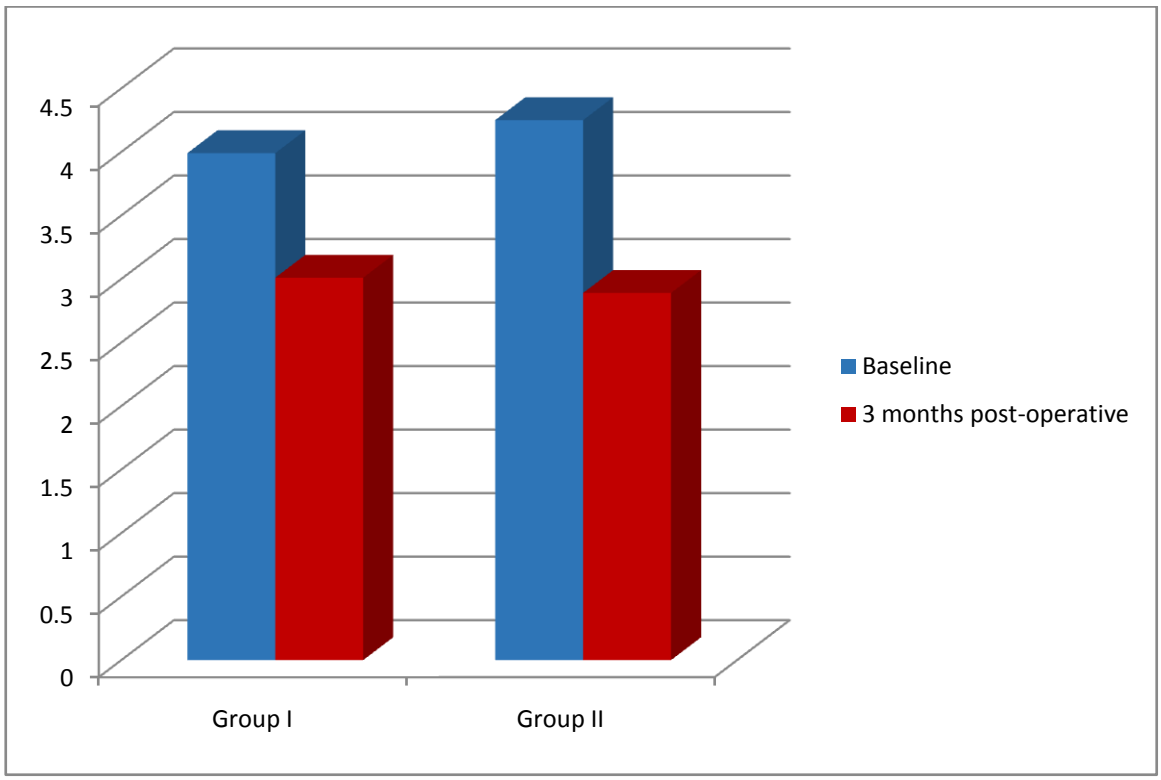
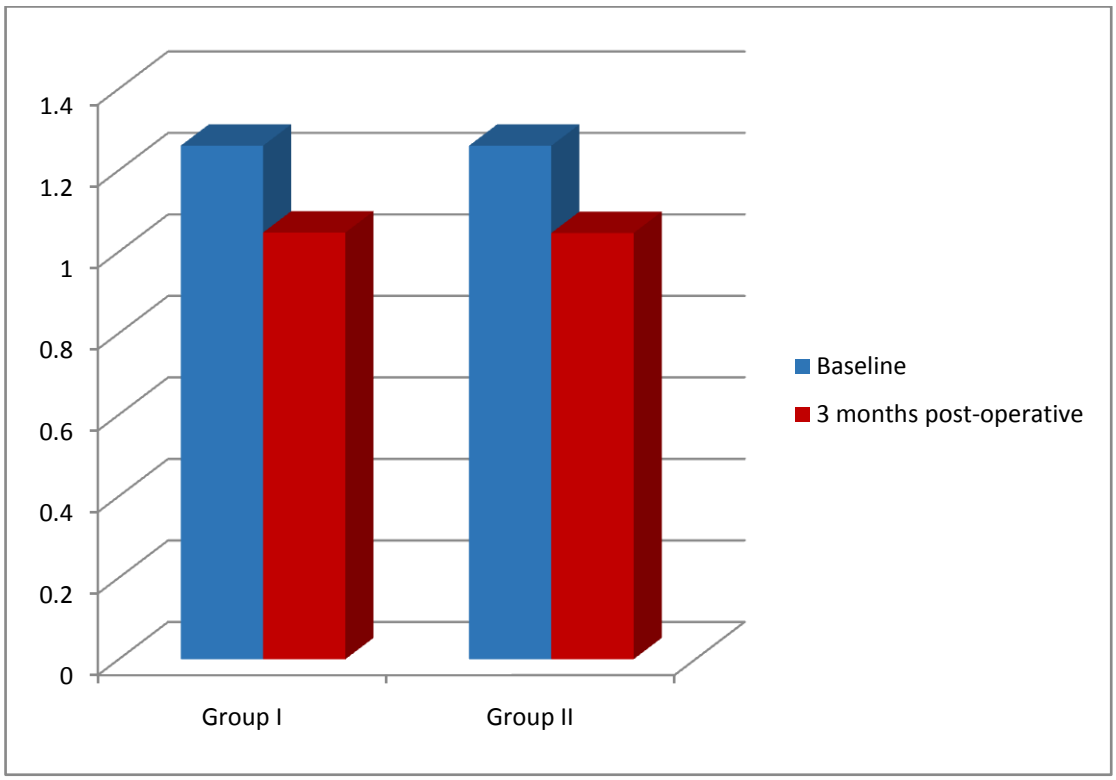


Figure 5 **: Comparison of osteopontin level between group I and group II**



Discussion

DISCUSSION

Periodontitis is an inflammatory disease of the supporting tissues surrounding the teeth caused by heterogenous group of microorganisms, resulting in progressive destruction of the periodontal ligament, which if left untreated will progress to alveolar bone destruction ultimately resulting in tooth loss ***(Marugame et al 2003)***[83].

The sine qua non of periodontal treatment is to preserve the dentition. Ideally, therapy should resolve inflammation, arrest disease progression, maintain aesthetics, maximize patient comfort, regenerate lost periodontium and create an environment that deters recurrent disease. To accomplish these goals both surgical and non-surgical procedures are employed. To date, the critical objective of periodontal therapy has been to halt disease progression. In this regard, clinical trials indicate that scaling and root planing can arrest periodontitis ***(Badersten et al 1987, Philstrom et al 1981, Philstrom et al 1984, AAP 1989)***[84,52,54,72] **.**

Numerous studies indicated mechanical instrumentation improved periodontal status ***(Listgarten et al 1978, Caffesse et al,1986)***[85,86] . After mechanical instrumentation, probing depth reduction results from gingival recession and a gain of clinical attachment ***(Hughes & Caffesse 1978, Proye et al 1982)***[42,43]. In general, the deepest sites demonstrated the greatest pocket reduction after instrumentation ***(Knowles 1979, Hammerle et al 1991)***[87,88]**.** Similarly, the magnitude of recession was related to initial probing depths and inflammatory status of the tissues. The most gingival shrinkage occurred interproximally. After scaling and root planing, investigators reported decreased gingival inflammation and less bleeding upon probing in patients with chronic periodontitis ***(Singletary et al 1982, Greenewell et al 1984, Lavanchy et al 1987)***[39,40,41]**.**

This study was conducted to evaluate the efficacy of homeopathy medicine as an adjunct to scaling and root planning in patients with chronic periodontitis. The medicine used here was *Calcarea fluor 30s.* Even though it has been used to evaluate the remineralization efficacy on artificial carious lesions on enamel the present study is the only study till date to access the effectiveness of calc-f tablets on periodontium.

In the present study, subjects with any acute or chronic systemic conditions like diabetes or inflammatory conditions like rheumatoid arthritis, cardiovascular diseases etc. had been excluded because these conditions can cause increased osteopontin level on their own, which may lead to confounding effect in the study.

Patients under medications like antibiotics, corticosteroids or anti-inflammatory drugs for the past 3 months and those underwent periodontal therapy within the past six months have also been excluded because these therapies can suppress the inflammatory process and may lead to confounding effect in the study.

Quantitative determination of osteopontin in the present study in both groups was done by double antibody sandwich ELISA method which is a very sensitive method for comparing osteopontin.

In the present study two groups with 10 patients each having chronic periodontitis were evaluated. In group I, the patients underwent scaling and root planning alone while in group II scaling and root planning was accompanied by systemic administration of calcarea fluor for 3 months. The clinical parameters like gingival bleeding index, plaque index, pocket probing depth and clinical attachment level were measured for each group at baseline and 3 months after therapy. Also comparison of osteopontin level was done between the two groups before and after therapy.

Bleeding on probing is a commonly used diagnostic criterion for periodontal diseases. A highly significant reduction was observed in both group I ($p = 0.00$) and group

II (p = 0.001) before and after therapy. The sustained reduction in bleeding on probing is of practical interest because the lack of bleeding is a reasonable prognosticator of periodontal health. Stability is anticipated when bleeding is reduced.

Plaque scores reflect the oral hygiene status of the patient than the disease severity. Plaque index in group I was 2.42 ± 0.23 at baseline which had decreased to .993 ± 0.275 , 3 months after therapy, which showed a statistically significant result. In group II, the plaque index was 2.45 ± 0.31 at baseline 1.048 ± 0.318 post-treatment, which also showed a statistically significant result. But there was no statistically significant difference between group I and group II after therapy.

Probing pocket depth measurements are of prime importance in evaluationg the success of a periodontal therapy. The probing pocket depth in group I was 3.66 ±1.06 at baseline and 2.64 ± 0.764 after therapy. This showed a statistically significant difference and the results are consistent with the studies done by ***Lindhe et al 1982***[56]. In their split mouth study comparing scaling and root planning to modified Widman flap, they found that the pocket depth reduced from 4.2 to 2.9mm in the SRP group. Numerous other studies by ***Philstrom et al. (1981, 1983,1984)***[52,53,54]***, Hill et al. (1981)***[55]***, Kerry et al. (1990)*** [63] also showed a statistically significant reduction in probing depth before and after scaling and root planing. The probing depth in group II was 3.84±0.62 at baseline and 2.091 ±0.723 after use of homeopathy medicine along with scaling and root planning. This result showed a statistically significant difference between group I and group II, 3 months after therapy (0.021).

Change in clinical attachment level denotes the status and condition of the tissues. The clinical attachment level in group I was 3.97 ±1.10 at baseline and 3.005 ± 0.971, 3 months after the phase I therapy. This shows a statistically significant reduction in attachment level which correlates with the studies conducted by ***Cobb 1996***[46]***, Becker et al***

1988[64], Isidor et al (1984,1985)[59,60], Isidor and Karring (1986)[61]. In group II the reduction was from 4.25 ±0.72 at baseline to 2.889 ± 0.645 after therapy. Though there was no statistically significant difference between group I and group II after therapy, the reduction was more evident in group II.

The present study showed significantly higher levels of osteopontin in group I (mean 1263.30ng/ml) at baseline as compared to 3 months after treatment (1047.2ng/ml) . Group II also showed almost the same values at baseline (1261.7ng/ml) and post-therapy (1046.7ng/ml). The result correlates with the findings of ***Hans and Mali* (*2012)[89]*** who observed statistically significant decrease in osteopontin level between baseline and 2 months after phase I therapy (153.09ng/ml, 91.52ng/ml). In their study assessment of osteopontin was done using Osteopontin enzyme immunometric assay kit whereas in the present study the analysis was done using sandwich type human osteopontin enzyme immunoassay kit. But there was no statistically significant difference between group I and group II in osteopontin level before and after therapy. The result of the present study was similar to the result obtained by ***Sharma and Pradeep (2007)[31]***. The highest mean gingival crevicular fluid level of OPN in periodontitis subjects in their study was 1575.01 ng/ml and after phase I therapy it decreased to 1194.80 ng/ml. The OPN level in plasma was 1273.21 ng/ml which decreased to 1051.68 ng/ml after phase I therapy.

The present study was conducted to evaluate if homeopathy treatment improved the results of phase I therapy in chronic periodontitis patients. Though plaque index, gingival bleeding index, CAL and osteopontin level showed no statistically significant difference, there was a significant difference in PPD between group I and group II. More research with a larger sample size may yield a better result and will help to evaluate the effectiveness of this homeopathy medicine in dental field.

Summary and Conclusion

SUMMARY AND CONCLUSION

The present study was conducted in order to evaluate the effectiveness of *calcarea fluor* as an adjunct to scaling and root planning in patients with chronic periodontitis.

A total of 20 patients with chronic periodontitis were selected and divided into two groups of 10 each. Group I patients were treated with scaling and root planning alone while group II patients were given *calcarea fluor* for 3 months after scaling and root planning. The clinical parameters and osteopontin analysis were done at baseline and 3 months after therapy and thevalues were subjected to statistical analysis.

The following conclusions were drawn from the study:

- The osteopontin levels are high in patients with chronic periodontitis and the value show appreciable reduction after phase I therapy.
- Even though clinical parameters show a considerable improvement after phase I therapy, there is no appreciable difference after use of *calcarea fluor* except for probing pocket depth.

In future, clinical interventional studies with larger sample size may be employed to further evaluate the efficacy of *calcarea fluor* as an adjunct for treating periodontal diseases and maintaining and improving oral health status of the patient.

Bibliography

BIBLIOGRAPHY

1. Frodge BD, Ebersole JL, Kryscio RJ, Thomas MV, Miller CS. Bone Remodeling Biomarkers of Periodontal Disease in Saliva. J Periodontol 2008;79:1913-19.
2. Teles R, Sakellari D, Teles F, Konstantinidis A, Kent R, Socransky S, et al . Relationships among Gingival Crevicular Fluid Biomarkers, Clinical Parameters of Periodontal Disease, and the Subgingival Microbiota. J Periodontol 2010;81:89-98.
3. Rodan GA. Osteopontin overview. Annals of New York Academic Sciences; 1995; 1-5. Department of Bone Biology and Osteoporosis Research, Merck Research Laboratories, West Point, Pennsylvania 19486, USA, Osteopontin: Role in Cell Signalling and Adhesion. Proceedings of a conference. New Brunswick, New Jersey, October 21-23, 1994.
4. Lessell C B. A Textbook of Dental Homeopathy Saffron Walden: The C W Daniel Company Ltd, 1995.
5. Kinane, D. F. (2001) Causation and pathogenesis of periodontal disease. *Periodontology 2000* **25**, 8-20.
6. Page, R. C. & Kornman, K. S. (1997) The pathogenesis of human periodontitis: an introduction. *Periodontology 2000* **14**, 9-11.
7. Philstrom, B. L., Michalowicz, B. S. & Johnson, N. W. (2005) Periodontal diseases. *Lancet* **366**, 1809-1820.
8. Armitage GC. Clinical evaluation of periodontal diseases. Periodontol 2000 1995;7:39-53.
9. Beck JD, Offenbacher S. Systemic effects of periodontitis: Epidemiology of periodontal disease and cardiovascular disease. J Periodontol 2005;76:2089-2100.
10. Genco RJ, Trevisan M, Wu T, Beck JD. Periodontal disease and risk of coronary heart disease. JAMA 2001; 285:40-41.

11. Seymour GJ, Ford PJ, Cullinan MP, Leishman S, Yamazaki K. Relationship between periodontal infections and systemic disease. Clin Microbiol Infect 2007; 13(Suppl. 4):3-10.
12. Mealey BL, Oates TW. Diabetes mellitus and periodontal diseases. J Periodontol 2006;77:1289-1303.
13. Taba M Jr, Kinney J, Kim AS, Giannobile WV. Diagnostic Biomarkers for Oral and Periodontal Diseases. Dent Clin North A. 2005;49:551-72.
14. Patarca R, Freeman GJ, Singh RP, Wei FY, Durfee T, Blattner F, Regnier DC, Kozak CA, Mock BA, Morse HC III. Structural and functional studies of the early T lymphocyte activation 1 (Eta -1) gene. Definition of a novel T-cell dependent response associated with genetic resistance to bacterial infection. J Exp Med 1989;170:145-61.
15. Standal T, Borset M, Sundan A. Role of osteopontin in adhesion , migration, cell survival and bone remodeling. Experimental oncology; October 2004.
16. Steitz SA, Speer MY, McKee MD, Liaw L, Almeida M, Yang H, Giachelli CM. Osteopontin inhibits mineral deposition and promotes regression of ectopic calcificatopn. Am J Pathol 2002; 161: 2035-46.
17. Hunter GK, Hauschka PV, Poole AR, Rosenberg LC, Goldberg HA. Nucleation and inhibition of hydroxyapatite formation by mineralized tissue proteins. Biochem J 1996; 317:59-64.
18. Pampena DA, Robertson KA, Litvinova O, Lajoie G, Goldberg HA, Hunter GK. Inhibition of hydroxyapatite formation by osteopontin phosphopeptides. Biochem J 2004; 378:1083-7.
19. Teitelbaum SL. Bone resorption by osteoclasts. Science 2000; 289:1504-8.

20. Rittling SR, Matsumoto HN, McKee MD, Nanci A, An XR, Novick KE, Kowalski AJ, Noda M, Denhardt DT. Mice lacking osteopontin show normal development and bone structure but display altered osteoclast formation in vitro. J Bone Miner Res 1998; 13:1101-11.
21. Liaw L, Birk DE, Ballas CB, Whitsitt JS, Davidson JM, Hogan BL. Altered wound healing in mice lacking a functional osteopontin gene (spp 1). J Clin Inves 1998;101:1468-78.
22. Ihara H, Denhardt DT, Furuya K, Yamashita T, Muguruma Y, Tsuji K, Hruska KA, Higashio K, Enomoto S, Nifuli A, Rittiling SR, Noda M. Parathyroid hormone0-induced bone resorption does not occur in the absence of osteopontin. J Biol Chem 2001;276:13065-71.
23. Yamate T, Mocharla H, Taguchi Y, Igietseme JU, Manolagas SC, Abe E. osteopntin expression by osteoclast and osteoblast progenitors in the murine bone marrow: demonstration of its requirement for osteoclastogenesis and its increase after ovariectomy. Endocrinology 1197; 138: 3047-55.
24. Ishii T, Ohshima S, Ishida T, Mima T, Tabunoki Y, Kobayashi H, Maeda M, Uede T, Liaw L, Kinoshita N, Kawase I, Saeki Y. osteopontina as a positive regulatoe in the osteoclastogenesis of arthritis. Biochem Biophys Res Commun 2004; 316:809-15.
25. Faccio R, Grano M, Colucci S, Zallone AZ, Quaranta V, Pelletier AJ. Activation of alpha v beta 3 integrin on human osteoclast-like cells stimulates adhesion and migration in response to osteopontin. Biochem Biophys Res Commun 1998; 249: 522-5.
26. Faccio R, Grano M, Colucci S,Villa A, Giannelli G, Quaranta V, Zallone A. Localization and possible role of two different alpha v beta 3 integrin conformations in resting and resorbing osteoclasts. J Cell Sci 2002;115:2919-29.

27. Chellaiah MA, Hruska KA. The integrin alpha (v) beta(3) and CD44 regulate the actions of osteopontin onosteoclast motility. Cacif Tissue Int 2003;72:197-205.
28. Tani-Ishii N, Tsunoda A, Umemoto T. osteopontin antisense deoxyoligonucleotides inhibit bone resorption by mouse osteoclasts in vitro. J Periodontal Res 1997;32:480-6.
29. Dodds RA, Connor JR, James IE, Rykaczewski EL, Applebaum E, Dul E, Gowen M. Human osteoclasts, not osteoblasts , deposit osteopontin into resoption surfaces:an in vitro and ex vivo study of remodeling bone. J Bone Miner Res 1995; 10:1666-80.
30. Merry K, Dodds R, Littlewood A, Gowen M. Expression of osteopontin mRNA by osteoclasts and osteoblasts in modeling adult human bone. J Cell Sci 1993;104:1013-20.
31. Sharma CG, Pradeep AR. Plasma and crevicular fluid osteopontin levels in periodontal health and disease. J Periodontal Res 2007;42:450-5.
32. Haffajee AD, Cugini MA, Dibart S, Smith C, Kent RL, Jr. & Socransky SS. (1997). The effect of SRP on the clinical and microbiological parameters of periodontal diseases. *Journal of Clinical Periodontology* **24**, 324-334.
33. Magnusson I, Lindhe J, Yoneyama T & Liljenberg B. (1984) Recolonization of a subgingival microbiota following scaling in deep pockets. *Journal of Clinical Periodontology* **11**, 193-207.
34. Badersten A, Nilvéus R & Egelberg J. (1981) Effect of nonsurgical periodontal therapy. I. Moderately advanced periodontitis. *Journal of Clinical Periodontology* **8**, 57-72.
35. Torfason T, Kiger R, Selvig KA & Egelberg J. (1979) Clinical improvement of gingival conditions following ultrasonic versus hand instrumentation of periodontal pockets. *Journal of Clinical Periodontology* **6**, 165-176.

36. Leon LE & Vogel RI. (1987) A comparison of the effectiveness of hand scaling and ultrasonic debridement in furcations as evaluated by differential dark-field microscopy. *Journal of Periodontology* **58**, 86-94.
37. Nyman S, Westfelt E, Sarhed G & Karring T. (1988) Role of "diseased" root cementum in healing following treatment of periodontal disease. A clinical study. *Journal of Clinical Periodontology* **15**, 464-468.
38. Oberholzer R & Rateitschak KH. (1996) Root cleaning or root smoothing. An in vivo study. *Journal of Clinical Periodontology* **23**, 326-330.
39. Singletary MM, Crawford JJ, Simpson DM. Dark-field microscopic monitoring of subgingival bacteria during periodontal therapy. J Periodontal 1982; 53:671-681.
40. Greenwell H, Bissada NF. Variations in subgingival microflora from healthy and intervention sites using probing depth and bactériologie identification criteria. J Periodontal 1984; 55:391-397.
41. Lavanchy D, Bickel M, Bachni P. The effect of plaque control after scaling and root planing on the subgingival microflora in human periodontitis. J Clin Periodontal 1987; 14:295-299.
42. Hughes TP & Caffesse RG. (1978) Gingival changes following scaling, root planning and oral hygiene. A biometric evaluation. *Journal of Periodontology* **49**, 245-252.
43. Proye M, Caton J & Polson A. (1982) Initial healing of periodontal pockets after a single episode of root planing monitored by controlled probing forces. *Journal of Periodontology* **53**, 296-301.
44. Morrison E, Ramfjord S, Burgett F, Nissle R, Shick R. The significance of gingivitis during the maintenance phase of periodontal treatment. *J Periodontol* 1982;53:31-34.
45. Ramfjord S, Morrison E, Burgett F, et al. Oral hygiene and maintenance of periodontal support. *J Periodontol* 1982;53:26-30.

46. Cobb CM. (1996) Non-surgical pocket therapy: mechanical. *Annals of Periodontology* **1**, 443-490.
47. Cobb CM. (2002) Clinical significance of non-surgical periodontal therapy: an evidence-based perspective of scaling and root planing. *Journal of Clinical Periodontology* **29**, 6-16.
48. Badersten A, Nilveus R, Egelberg J. Effect of non-surgical periodontal therapy. II. Severely advanced Periodontitis.J Clin Periodontal 1984; 11:63-76.
49. Claffey N & Egelberg J. (1995) Clinical indicators of probing attachment loss following initial periodontal treatment in advanced periodontitis patients. *Journal of Clinical Periodontology* **22**, 690-696.
50. Axelsson P & Lindhe J. (1981) The significance of maintenance care in the treatment of periodontal disease. *Journal of Clinical Periodontology* **8**, 281-294.
51. Renvert S & Persson GR. (2004) Supportive periodontal therapy. *Periodontology 2000* **36**, 179-195.
52. Philstrom B, Ortiz-Campos C, McHugh R. A randomized four-year study of periodontal therapy. *J Periodontal* 1981;52:227-242.
53. Philstrom B, McHugh R, Oliphant T, Ortiz-Campos C. Comparison of surgical and nonsurgical treatment of periodontal disease. A review of current studies and additional results after 6 1/2 years. *J Clin Periodontol* 1983;10:524-541.
54. Philstrom B, Oliphant T, McHugh R. Molar and nonmolar teeth compared over 6-1/2 years following two methods of periodontal therapy. *J' Periodontol* 1984;55:499-504.
55. Hill R, Ramfjord S, Morrison E, et al. Four types of periodontal treatment compared over two years. *J Periodontol* 1981;52:655-662.

56. Lindhe J, Westfelt E, Nyman S, Socransky S, Heijl L, Bratthall G. Healing following surgical/non-surgical treatment of periodontal disease. A clinical study. *J Clin Periodontol* 1982A;9:115-128.
57. Lindhe J, Nyman S, Westfelt E, et al. "Critical probing depths" in periodontal therapy. *Compendium Contin Educ Dent* 1982B;3:421-30.
58. Lindhe J, Westfelt E, Nyman S, Socransky S, Haffajee A. Long-term effect of surgical/non-surgical treatment of periodontal disease. *J Clin Periodontol* 1984; 11:448-458.
59. Isidor F, Karring T, Attstrom R. The effect of root planing as compared to that of surgical treatment. *J Clin Periodontol* 1984; 11:669-681.
60. Isidor F, Attstrom R, Karring T. Regeneration of alveolar bone following surgical and nonsurgical periodontal treatment. *J Clin Periodontol* 1985;12:687-696.
61. Isidor F, Karring T. Long-term effect of surgical and nonsurgical periodontal treatment. A 5 year clinical study. *J Periodont Res* 1986;21: 462-172.
62. Becker W, Becker B, Ochsenbein C, Kerry G, Caffesse R. A longitudinal study comparing scaling, osseous surgery and modified Widman procedures. Results after one year. *J Periodontol* 1988;59:351-365.
63. Kerry G, Becker W, Morrison E, Ochsenbein B, Becker B, Caffesse R. Three modalities of periodontal therapy: I. 5 year final results. *J Dent Res* 1990;69(Spec. Issue):246(Abstr. 1103).
64. Becker B, Becker W, Caffesse R, Kerry G, Ochsenbein C, Morrison E. Three modalities of periodontal therapy: II. 5 year final results. *J Dent Res* 1990;69:219.
65. Kaldahl W, Kalkwarf K, Patil K, Dyer J, Bates R. Evaluation of four modalities of periodontal therapy. *J Periodontol* 1988;59:783-793.

66. Kaldahl W, Kalkwarf K, Patil K, Molvar M. Responses of four tooth and site groupings to periodontal therapy. *J Periodontol* 1990;61:173-179.
67. Kalkwarf K, Kaldahl W, Patil K. Patient preference regarding 4 types of periodontal therapy following 3 years of maintenance follow-up. *J Clin Periodontol* 1992;19:788-793.
68. Cercek JF, Kiger RD, Garret S, Egelberg J. Relative effects of plaque control and instrumentation on the clinical parameters of human periodontal disease. Clin Periodontal 1983; 10:46-56.
69. Badersten A, Nilveus R, Egelberg J. Effect of nonsurgical therapy. III. Single versus repeated instrumentation. *J Clin Periodontol* 1984B;11: 114-124.
70. Badersten A, Nilveus R, Egelberg J. Effect of nonsurgical periodontal therapy. IV. Operator variability. *J Clin Periodontol* 1985A;12:190-200.
71. Badersten A, Nilveus R, Egelberg J. Effects of nonsurgical periodontal therapy. V. Patterns of probing attachment loss in non-responding sites. J *Clin Periodontol* 1985B;12:270-282.
72. Non-surgical periodontal treatment: consensus statement. In: Proceedings of the World Workshop in Clinical Periodontics. Chicago: American Academy of Periodontology; 1989: II-13–II-7.
73. Discovering Homeopathy: Medicine for the 21st Century by Dana Ullman (North Atlantic, 1991).
74. Peter Darby. How homeopathy can be used in dental practice. Dental Nursing 2011; Vol 7: No 11.
75. Linde K, Jonas WB, Melchart D et al (1994) Critical review and meta-analysis of serial agitated dilutions in experimental toxicology. *Hum Exp Toxicol* **13**: 481–92.

76. Belon P, Cumps J, Ennis M et al (2004) Histamine dilutions modulate basophil activation. *Inflamm Res* **53**: 181–8.

77. Witt CM, Bluth M, Albrecht H et al (2007) The in vitro evidence for an effect of high homeopathic potencies—A systematic review of the literature. *Complement Ther Med* **15**: 128–38.

78. Boericke W. Calcarea Fluorica. In: Boericke New Manual of Homoeopathic Materia Medica and Repertory. 2nd ed. New Delhi: Jain Publishers; 2000. p. 149-51.

79. Kalpana Bansal, Nidhi Balhara, Mohita Marwaha. Remineralizing efficacy of Calcarea Fluorica tablets on the artificial carious enamel lesions using scanning electron microscope and surface microhardness testing: *In vivo* study. Indian Journal of Dental Research, 25(6), 2014;25:777-82.

80. Silness J, Loe H. Periodontal disease in pregnancy. II. Correlation between oral hygiene and periodontal conditions. Acta Odontol Scand. 1964; 22: 121-35.

81. Ainamo J, Bay.I. Problems and proposals for recording gingivitis and *plaque. Int. Dent. J* 1975; 25: 229-235.

82. Carranza's *Clinical Periodontology*. Eleventh edition 2012.

83. Marugame T, Hayasaki H, Lee K, Eguchi H, Matsumoto S. Alveolar bone loss associated with glucose tolerance. Japanese men. Diabet Med 2003; 20:746-751.

84. Badersten A, Nilveus R, Egelberg J. 4 year observations of basic periodontal therapy. J Clin Periodontol 1987; 14:438-444.

85. Listgarten MA, Lindhe J, Hellden LB. Effect of tetracycline and/or scaling on human periodontal disease. J Clin Periodontal 1978; 5:246-271.

86. Caffesse RG, Sweeney PL, Smith BA. Scaling and root planing with and without periodontal flap surgery. / Clin Periodontal 1986; 13:205- 211.

87. Knowles JW, Burgett FG, Nissle RR, et al. Results of periodontal treatment related to pocket depth and attachment level. J Periodontal 1979; 50:225-233

88. Hammerle CHF, Joss A, Lang NP. Short term effects of initial periodontal therapy (hygienic phase). J Clin Periodontol 1991; 18:233- 239.

89. Hans S, Mali AM. Estimation and comparison of osteopontin levels in plasma in subjects with healthy periodontium and generalized chronic periodontitis and its assessment after scaling and root planing. J Indian Soc Periodontol 2012;16:354-7.

Annexures

Annexure 1 : Participant information sheet (English)

Participant information sheet

Investigator : Anju M K Guide : K. Malathi, MDS

Title : CLINICAL AND BIOCHEMICAL EVALUATION OF EFFICACY OF CALCIUM FLUORIDE AS AN ADJUNCT TO NON-SURGICAL PERIODONTAL THERAPY – A RANDOMIZED CLINICAL TRIAL

Name of the research institution : Tamilnadu Government Dental College and Hospital, Chennai

The investigator, Dr. ANJU M K under the guidance of Dr. K MALATHI, MDS and Dr. V.S. PARTHIBAN, DHMS is conducting a study as titled above with aim to do an evaluation of calcium fluoride usage along with scaling and root planning in chronic periodontitis.

1. **Procedure : the following examinations and investigations will be done for you.**

- Intraoral examination, Extraoral examination
- Blood test– 7ml of blood will be drawn from your hand
- X-ray will be taken for the diseased site
- Model of your teeth will be prepared by taking alginate impression
- Deposits on your teethwith ultrasonic scaler and hand instrument. Homeopathy drug may or may not be given to the patient. Further clinical evaluation will be performed at baseline, 3 months and 6 months after the procedure. 2ml of blood will be drawn again at 6 months.

2. **Risk of participation:**

- Patients may be allergic to LA or the material used in the study.
- Patient may experience pain, discomfort, swelling following the procedure.

3. **Benefits of participation:**

Patients will be treated for improving the periodontal status and minimizing alveolar bone loss.

4. **Confidentiality :**

The identity of the patients participating in the research will be kept confidential throughout the study. In the event of any publication or presentation resulting from the research, no personally identifiable information will be shared.

5. **Participants right :**

Taking part in the study is voluntary. You are free to decide whether to participate in the study or to withdraw at any time; your decision will not result in any loss of benefits to which you are otherwise entitled. The results of this study will be intimated to you at the end of the study period or during the study if anything is found abnormal which may aid in the management or treatment.

6. **Compensation: Nil**

7. **Contacts:**

For queries related to the study:
Primary Investigator: Dr.ANJU M K
Contact Details: PG Student
Department of Periodontics
Tamilnadu Govt. Dental College & Hospital
Chennai-600 003.
Phone number:7200909002

Contact details regarding rights of the participant:
Dr. B. Saravanan, MDS,PhD,
The Chairperson,
Institutional Ethical committee
Tamilnadu Govt. Dental College & Hospital, Chennai-600 003.

Annexure 2 : Participant information sheet (Tamil)

ஆராய்ச்சி பற்றிய தகவல் படிவம்

ஆராய்ச்சி மேற்கொள்பவர்
மருத்துவர் எம்.கே.அஞ்சு

வழிநடத்துபவர்
மருத்துவர்.கே.மாலதி

ஆராய்ச்சியின் தலைப்பு

அறுவை சிகிச்சையில்லா ஈறு அழற்சி மருத்துவம் முறையுடன் ஹோமியோபதி மருந்துகளைக் கூடுதலாக பயன்படுத்தும்போது ஏற்படும் நன்மைகள் மற்றும் அவற்றின் செயல்திறன்- ஒரு மருத்துவ மற்றும் உயிர்வேதியியல் ஒப்பீட்டு மதிப்பீடு

செய்முறை

கீழ்கண்ட ஆய்வுகள்/ பரிசோதனைகள் உங்களுக்காக செய்யப்படும்.

- வாய் பரிசோதனை
- உட்புறம்
- வெளிப்புறம்
- வழக்கமான இரத்தப் பரிசோதனை
- உங்களின் கையிலிருந்து இரத்தப் பரிசோதனைக்காக 7 மில்லி அளவு இரத்தம் எடுக்கப்படும்.
- நோயுற்ற பகுதியின் ஊடுகதிர் படம்.
- ஒவ்வாமை ஏற்படுகிறதா என்பதை தெரிந்துகொள்ள 0.5மி.லி 2% லிக்னோகெயின் மயக்க மருந்து உங்களின் கையில் பரிசோதனைக்காக செலுத்தப்படும். பின்பு நோயுற்ற பகுதியில் மயக்க மருந்து கொடுக்கப்படும்.
- அல்ட்ரா சோனிக் ஸ்கேலர் மற்றும் கைக்கருவிகள் பயன்படுத்தி பல் மற்றும் பல்லின் வேர் சுத்தம் செய்யப்படும். உப்புநீர் கொண்டு நோயுற்ற பகுதி சுத்தம் செய்யப்படும்.
- சிகிச்சையுடன் கூடுதலாக ஹோமியோபதி மருந்துகள் பயன்படுத்தப்படும்

பங்கேற்பதினால் வரக்கூடிய பக்க விளைவுகள்: பங்கேற்பதினால் மிக அரிதாக ஏற்படும் ஒவ்வாமை ஏற்பட்டாலும் அதற்கு தேவையான மருந்துகளும் மருத்துவமும் வழங்கப்படும்.

பங்கேற்பதினால் விளையும் நன்மைகள்: உங்கள் ஈறு அழற்சி நோய்க்கு சிகிச்சை அளிக்கப்படும்.

இரகசிய காப்பு உங்களைப் பற்றிய குறிப்புகள் பிறர் அறியா வண்ணம் ஆராய்ச்சி முடியும் வரை இரகசியமாக பாதுகாக்கப்படும். அதை வெளிப்படுத்தும் நேரங்களில் எந்த தனி அடையாளங்களும் வெளிப்பட வாய்ப்பு கிடையாது.

தன்னார்வ பங்கேற்பு இந்த ஆராய்ச்சியில் பங்குபெறுவது தங்களின் தனிப்பட்ட முடிவு மற்றும் இந்த ஆராய்ச்சியில் இருந்து நீங்கள் எப்போது வேண்டுமானாலும் விலகிக்கொள்ளலாம். தங்களின் இந்த தீர் முடிவு உங்களுக்கோ அல்லது ஆராய்ச்சியாளருக்கோ எந்தவித பாதிப்பும் ஏற்படுத்தாது என்பதை தெரியப்படுத்துகிறோம்.

நோயாளியின் பெயர் **கையொப்பம்/ கைரேகை**

ஆராய்ச்சி தொடர்புடைய தகவல்களுக்கு
மரு.எம்.கே.அஞ்சு,
தனம் பெண்கள் கிளினி.
84/2, பூசாலிகெங்குரெட்டி தெரு,
எழும்பூர், சென்னை-600 008.

பங்கேற்பாளரின் உரிமை தொடர்புடைய தகவல்களுக்கு: மரு.பி.சரவணன்
தலைவர், நிறுவன நெறிமுறைகள் குழு,
தமிழ்நாடு அரசு பல் மருத்துவக் கல்லூரி
மற்றும் மருத்துவமனை, சென்னை-3.

Annexure 3 : Informed Consent Form (English)

Informed Consent Form

CLINICAL AND BIOCHEMICAL EVALUATION OF EFFICACY OF CALCIUM FLUORIDE AS AN ADJUNCT TO NON-SURGICAL PERIODONTAL THERAPY – A RANDOMIZED CLINICAL TRIAL

Participant ID No:

"I have read the foregoing information, or it has been read to me. I have had the opportunity to ask questions about it and any questions I have asked have been answered to my satisfaction. I consent voluntarily to participate as a participant in this study and understand that I have the right to withdraw from the study at any time without in any way it affecting my further medical care."

_____	_____	_____
Date	Name of the participant	Signature/thumb impression Of the participant

[The literate witness selected by the participant must sign the informed consent form. The witness should not have any relationship with the research team; If the participant doesn't want to disclose his / her participation details to others, in view of respecting the wishes of the participant, he / she can be allowed to waive from the witness procedure (This is applicable to literate participant ONLY). This should be documented by the study staff by getting signature from the prospective participant]

_____

_____

_____

"I have witnessed the accurate reading of the consent form to the potential participant and the individual has had opportunity to ask questions. I confirm that the individual has given consent freely"

_____	_____	_____
Date	Name of the witness	Signature of the witness

_____	_____	_____
Date	Name of the interviewer	Signature of the interviewer

Annexure 4 : Informed Consent Form (Tamil)

ஆராய்ச்சி ஒப்புதல் படிவம்

ஆராய்ச்சியின் தலைப்பு

அறுவை சிகிச்சையில்லா ஈறு அழற்சி மருத்துவம் முறையுடன் ஹோமியோபதி மருந்துகளைக் கூடுதலாக பயன்படுத்தும்போது ஏற்படும் நன்மைகள் மற்றும் அவற்றின் செயல்திறன்- ஒரு மருத்துவ மற்றும் உயிர்வேதியியல் ஒப்பீட்டு மதிப்பீடு

பெயர் நோயாளி எண்

வயது/ பால் ஆராய்ச்சி சேர்க்கை எண்

முகவரி

தொலைபேசி

நான் ..... வயது ..... என்னுடைய சய நினைவுடனும் மற்றும் முழு சுதந்திரத்துடனும் இந்த மருத்துவ ஆராய்ச்சியில் என்னை சேர்த்துக்கொள்ள ஒப்புதல் அளிக்கிறேன்.

கீழ்காணப்படும் நிபந்தனைகளுக்கு நான் சம்மதிக்கிறேன்.

நான் இந்த ஆராய்ச்சியின் நோக்கம் மற்றும் செயல்முறைகள் பற்றி முழுமையாக தெரிவிக்கப்பட்டுள்ளேன்.

இந்த பரிசோதனைக்காக பற்களை சுத்தம் செய்ய வேண்டியுள்ளதாக அறிகிறேன்.

சிகிச்சையின் போது ஹோமியோபதி மருந்துகளும் கூடுதலாக பயன்படுத்தப்படுவதாக அறிகிறேன்.

என் உடல் நலம் பாதிக்கப்பட்டாலோ அல்லது எதிர்பாராத வழக்கத்திற்கு மாறான நோய்குறிகள் தென்பட்டாலோ அதற்கு சிகிச்சை பெற்றுக்கொள்வதற்கும் முழு உரிமை உள்ளதாக அறிகிறேன்.

நான் ஏற்கனவே உட்கொண்ட மற்றும் உட்கொள்கின்ற மருந்துகளின் விபரங்களை ஆராய்ச்சியாளரிடம் தெரிவித்துள்ளேன்.

என் மருத்துவ குறிப்பேடுகளை இந்த ஆராய்ச்சியில் பயன்படுத்திக்கொள்ள சம்மதிக்கிறேன். இந்த ஆராய்ச்சி மையமும் ஆராய்ச்சியாளரும் என்னுடைய விபரங்கள் அனைத்தையும் இரகசியமாக வைப்பதாக அறிகிறேன்.

.....	.....	.....
நோயாளியின் பெயர்	கையொப்பம்	தேதி
.....	.....	.....
ஆராய்ச்சியாளர் பெயர்	கையொப்பம்	தேதி

Annexure 5 : Proforma

PROFORMA FOR TREATMENT GROUP

Date : OP No.: S.No.:

Name : Age : Sex:

Occupation : Income :

Address : Phone Number :

CHIEF COMPLAINTS AND DURATION:

HISTORY OF PRESENT ILLNESS:

PAST MEDICAL HISTORY:

PAST DENTAL HISTORY:

FAMILY HISTORY :

PERSONAL HISTORY :

a) Oral Hygiene Practices :

b) Habits :

c) Menstrual History :

d) Menopause :

e) H/o. Stress Factor :

GENERAL EXAMINATION

a) Extra-Oral Examination

b) Examination of Lymphnodes

INTRA-ORAL EXAMINATION WITH CLINICAL FINDINGS:

Buccal mucosa:

Vestibule:

Hard palate:

Soft palate:

Tonsils:

Tongue:

Floor of the mouth:

Teeth:

Decayed

Missed

Filled teeth

Gingiva:

Plaque index

18	17	16	15	14	13	12	11	21	22	23	24	25	26	27	28
48	47	46	45	44	43	42	41	31	32	33	34	35	36	37	38

Bleeding Index

18	17	16	15	14	13	12	11	21	22	23	24	25	26	27	28
48	47	46	45	44	43	42	41	31	32	33	34	35	36	37	38

Probing depth and attachment level in millimeter:

Maxillary:

CAL																
PPD																
	18	17	16	15	14	13	12	11	21	22	23	24	25	26	27	28
PPD																
CAL																

Mandibular:

CAL																
PPD																
	18	17	16	15	14	13	12	11	21	22	23	24	25	26	27	28
PPD																
CAL																

Investigations:

1. Biochemical / Haematological Investigation :

   Hb count

   Total Leucocyte Count

   Differential Leucocyte Count

   Bleeding Time

   Clotting Time

   Random Blood Sugar

2. Osteopontin level

3. Others :

   Blood Pressure :

   Test Dose for L.A:

RADIOGRAPHIC EVALUATION

Intra-Oral Periapical Radiograph/Orthopantomogram (IOPA/OPG)

PROVISIONAL DIAGNOSIS

PROGNOSIS

TREATMENT PLAN

FITNESS FOR TREATMENT

TREATMENT DONE

DATE : PROCEDURE : SIGNATURE :

MAINTENANCE PHASE

EVALUATION AFTER - 3 MONTH

Osteopontine level :

Gingiva

Plaque index

18	17	16	15	14	13	12	11	21	22	23	24	25	26	27	28
48	47	46	45	44	43	42	41	31	32	33	34	35	36	37	38

Bleeding index

18	17	16	15	14	13	12	11	21	22	23	24	25	26	27	28
48	47	46	45	44	43	42	41	31	32	33	34	35	36	37	38

Probing depth and attachment loss in millimeter:

Maxillary:

CAL																
PPD																
	18	17	16	15	14	13	12	11	21	22	23	24	25	26	27	28
PPD																
CAL																

Mandibular

CAL																
PPD																
	48	47	46	45	44	43	42	41	31	32	33	34	35	36	37	38
PPD																
CAL																

SIGNATURE OF THE P.G. SIGNATURE OF THE PROFESSOR

DATE: